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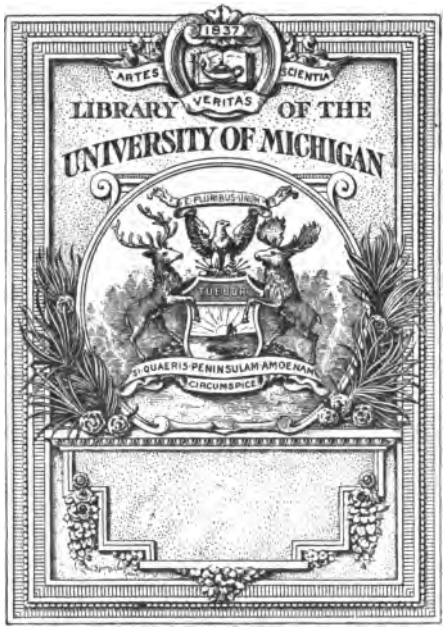
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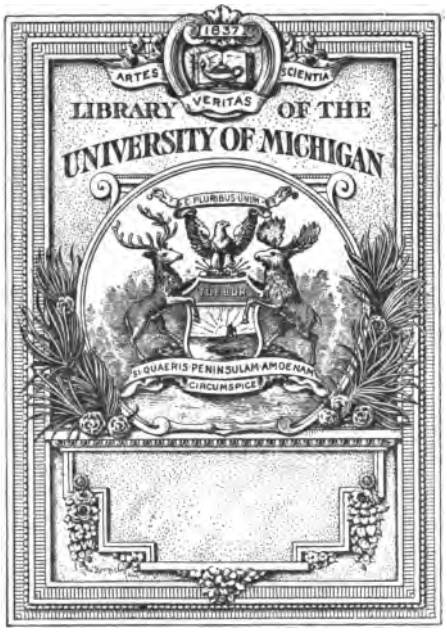
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THE

WESTMINSTER HOSPITAL REPORTS.

EDITED BY

GEORGE COWELL, F.R.C.S.

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ORIGINAL ARTICLES.

ON SOME OF THE PARALYSES OF CHILDHOOD.*

By Dr. DONKIN.

Impairment or loss of motor power in infants and young children may arise, as in adults, from lesion in any part of the motor tract, whether cerebral, spinal, neural, or neuro-muscular. The paralyses, however, which are due to chronic degeneration in brain or cord are mostly without their counterparts in early life, while others common to all periods, such as many of those caused by tumours and abscesses or by traumatic and other extraneous lesions of the nerves and nerve centres, have no clinical or pathological characters special to childhood.

Infantile Hemiplegia.

Paralysis on one side of the body either complete or partial in extent or degree and arising from lesion or defect of the cerebral motor tract is often met with in early childhood, and mainly differs from the classical hemiplegia of adults in being less often of that typical form, marked by involvement of the arm leg face and tongue, which results from lesions in or near the interna capsule, and on being much more frequently accompanied both by initial and subsequent convulsions. The reason for these differences lies in the rarity in childhood of hæmorrhage or thrombotic softening in the basal ganglia and in the more frequent situation of the lesion over the motor surface of the brain.

One of the chief known causes of hemiplegia in infants is *meningeal hæmorrhage* which however from its irregular distri-

* This paper is based on several clinical lectures chiefly on cases recorded in the writer's hospital note-books.

bution over the surface of the convolutions frequently produces bilateral symptoms. It is mostly traumatic and occurs at or soon after birth, owing to protracted labour or the use of instruments. In this context I may perhaps mention a case of hemiplegia with rigidity of the right arm and leg in a child of seven years old who was suffering from well-marked purpura hæmorrhagica. The paralysis improved and the diagnosis of superficial hæmorrhage seemed very probable. *Tumours* in the motor tract, whether in the cortex or lower down, *localised meningitis*, vascular changes from *thrombosis*, *embolism*, or *cerebral hæmorrhage*, and, rarely, *abscesses* of the brain, are also established causes, and *imperfect development of the motor cortex*, either congenital or sequent as atrophy on meningeal hæmorrhage or other trauma, has been several times observed.

Owing to the rarity of recent necropsies in hemiplegia in children the causes in numerous cases are matter of conjecture. Tumours, especially tubercular and cortical, are doubtless the origin of some cases otherwise at first unexplained, as was evidenced by the case of a boy of two years old under my care in hospital with left hemiplegia beginning with convulsions who was re-admitted after a year with recurrent convulsions and the signs of meningitis. At the post-mortem we found small caseous masses of tubercle in the upper Rolandic region on the right side and recent tubercular meningitis. Arterial embolism is a well-recognised though not frequent cause of hemiplegia in children, and may sometimes be confidently diagnosed in recent endocarditis or conjectured in some cases of marked stasis in the pulmonary circulation. I have notes of a case of rheumatic fever with endocarditis and chorea where sudden hemiplegia occurring first on the right side with aphasia and soon after on the left allowed no doubt of the diagnosis of double embolism. Hæmorrhage other than meningeal is but rarely proved in childhood, but it may occur in syphilitic disease of vessels, or in a growing tumour, and either hæmorrhage or thrombosis is the possible explanation of those cases of hemiplegia which take place during or soon after measles, scarlatina, and other acute infectious diseases. Among other examples of this I have seen a typical hemiplegia of capsular origin occurring three weeks after the onset of severe measles in a child of fifteen months; there was some rigidity, but all symptoms disappeared in four months.

Thrombosis of the right middle cerebral artery without heart disease or embolism was found by Dr. Abercrombie in a case of sudden left hemiplegia in a boy of six years old with diphtheria, and thrombosis of the vessels from syphilitic disease has frequently been reported. The sudden hemiplegia sometimes occurring in whooping-cough may be due to hæmorrhage from increased vascular pressure with or without anatomical change in the walls of the vessels. Many cases of infantile hemiplegia with convulsions may probably be explained, according to the ingenious and reasonable theory of Dr. Gowers (who argues from the frequent sinus-thrombosis of infancy), by the occurrence of thrombosis in cortical veins; and the hypothesis of Strümpell who reasons from the sclerosed and atrophied patches not seldom seen in the young brain and quotes that class of hemiplegias which begin, as infantile spinal paralysis often does, with febrile symptoms, that primary localised inflammation of the grey matter may cause cerebral palsy, is not to be disregarded although lacking anatomical proof.

It is perhaps possible, therefore, to diagnose the cause of hemiplegia in a few instances and to guess at it in others from its mode of onset; but seeing that in adult cases, with much more definite pathology, we frequently mistake between hæmorrhage, thrombosis, and embolism, I do not consider this question of much practical value. Clinically, infantile hemiplegia begins in most cases with convulsions, generally of the unilateral type, and loss of consciousness. Rigidity of the paralysed limbs as a rule sets in very soon, the joints of the upper extremity being most often flexed and the foot in the position of equino-varus. Convulsions both unilateral and general tend to recur; they are in many cases strictly epileptic and may remain after the paralytic symptoms have largely or quite disappeared. Mental deficiency of various degrees is very frequent, especially in the earliest and congenital cases, as also is marked abnormality in the shape and size of the skull, many of these infants being micro-cephalic. After a more or less lengthened period wasting of the rigid and paralysed limbs frequently sets in, and there may be marked surface coldness and venous congestion. The growth of the limbs is also often arrested. In cases other than congenital sudden onset is accompanied sometimes by marked pyrexia without further specific signs and, as we have seen, the attack may

arise in the course or sequel of many acute diseases. I have several times seen true aphasia in quite young children, in one case at the age of two years. Left hemiplegia with aphasia stated by some and supposed by others to be more frequent in children than in adults I have not yet observed. Fine tremor or ampler spasms on movement of the affected limbs, and the constant movements generally called "athetosis" are not infrequently seen. These latter movements, slow, irregular, and unlike either choreic or convulsive spasm, affect the upper extremity and mostly the fingers which are constantly working in a vermiform manner. They have also been inaccurately named "post-hemiplegic chorea," and far more appropriately, as preventing confusion with other disorders, "mobile spasm." In adults this symptom has generally been found in connection with softening in or near the optic thalamus, but it appears certain from general pathological knowledge that the various forms of mobile spasm with hemiplegia may be due, like the chronic rigidities, to lesions in any position which interfere with the fibres of the pyramidal, or motor, tract.

The *prognosis* in hemiplegia depends largely on the diagnosis of the cause, which, we have seen, is often of much difficulty. Congenital cases occurring with convulsions mostly involve idiocy, and frequent convulsions in any case may pass into epilepsy. Slight congenital cases however may improve or perhaps recover, and I have seen at least two non-congenital cases recover where convulsions had been frequent and rigidity marked. Frequent convulsions with monoplegiæ point to cortical lesion which, being often tubercular, may be ultimately followed by meningitis. The typical form of capsular hemiplegia is both as to its causes and prognosis similar to that which affects adults, and the forecast is here more favourable than in the larger class of cases due to affection of the cortex, for the lesion may be but small, and the mental faculties little if at all impaired. In acute cases, prolonged apathy and coma are of the worst augury. I may mention here that the temporary paralysis of the limbs seen after many and severe epileptic attacks does not occasion prolonged difficulty either in diagnosis or prognosis.

It follows from the prevailing permanency of these affections, from whatever cause arising, that the field of *treatment* is very small. It is rare that medical aid is at hand before the initial

convulsions are over, but, when possible, these should be checked, without waiting to make a diagnosis, according to the methods proper to the treatment of convulsions generally. The child should also be kept perfectly quiet, and bromide of ammonium or potassium should be given. There also is no objection to ordering iodide of potassium for some weeks, even in the absence of positive evidence of syphilis, for morbid processes due to syphilitic vascular disease may possibly thus be checked. Contracture may be treated by frequent shampooing of the limbs, but rarely, in my experience, with more success than by the probably useless faradic current. In two cases of mine of doubtful origin which made marked improvement no medicinal or manipulative treatment was employed.

Spastic Paralysis.

There is doubtless a small class of cases in infants and young children reminding the observer at first sight of that now well-known disease of adults which owing to the affection being chiefly or wholly, at least in the early stages, confined to the legs is described under the name of "spastic paraplegia." The chief clinical characteristics of this malady in adults are loss of power accompanied by startings and subsequently by rigidity of the limbs, increased knee-jerks, and ankle-clonus, while there is usually no wasting, affections of the sphincters, or impaired sensibility. These phenomena are frequent enough as a sequel of myelitis with descending degeneration of the lateral columns of the cord, but when occurring in an apparently pure and idiopathic form their pathology is at present uncertain. Some cases, progressive or stationary, are referred by many to a primary lateral sclerosis, while others, regressive or recovering, are sometimes classed with functional disorders. It is in my opinion to be regretted that the cases now to be considered should ever have been described under the name given to the adult affection; for, as we shall see, in but few of them are the symptoms of paraplegic distribution only, and it is not in the cord but rather in the brain that we must look for the underlying lesion.

Spastic paralysis in childhood, not only in its narrower but also in its wider sense, must be regarded as due to lesion or dis-

turbance of function in the cerebral motor tract, and is thus fittingly considered in connection with infantile hemiplegia.

Hemiplegia, as we have already seen, is most often accompanied by chronic spasm in infancy, and one at least of its causes, namely, meningeal hæmorrhage, is often productive of bilateral symptoms. It is known both from clinical and post-mortem evidence that many cases of congenital or very early spastic paralysis on one or both sides of the body are due to cortical injuries and wasting of the motor convolutions sequent on the lesion. Further, it is established by recorded cases* that the same symptoms may result from imperfect development of the motor convolutions on one or both sides. These considerations have an important bearing on many of the spastic paralyses of infancy including those which have been named "spastic paraplegia." Post-mortem examinations of the least complicated cases are few. I have myself seen but one in a markedly micro-cephalic idiot of nearly three years old where the motor convolutions were strikingly small and abnormal in appearance, and much regret that owing to the destruction of the specimen before a full description could be written I cannot record the case in detail.

Spastic paralysis, other than hemiplegic, in infancy is generally of congenital or very early origin; the arms mostly, and the trunk muscles sometimes, are affected as well as the legs. There is frequently marked wasting of the body and anæsthesia of varying degree, and, owing doubtless to cerebral causation, the sphincters are often spontaneously relaxed. Micro-cephaly and congenital idiocy are frequent in all grades, and also convulsions which in non-congenital cases are often the first noticed symptom and may be succeeded by mental disorder. The large majority of the whole and nearly all the congenital cases give evidence of mental deficiency. Strabismus, nystagmus, marked tremors or movement, and "mobile spasms" of the arms as well as the hands, have each been observed in one or other of my own cases. The knee-jerk and other "deep reflexes" are usually much increased, but the rigidity is often so great as to prevent the phenomenon of ankle-clonus. In the slighter cases when standing or walking is possible the rigidity is still conspicuous; the gait is hopping, the thighs adducted, the feet

* See Sharkey's "Lectures on Spasm in Chronic Nerve Disease," Churchill, 1886; and Ross's case in "Brain," vol. i., p. 477.

inverted, and the pointed toes frequently catch the ground. Most cases however are from the first unable to stand, and the rigidity is excessive, the legs often crossing one another. Sometimes the limbs are all in rigid flexion; in others the legs are extended while the arms are flexed.

The difference in the extent of the paralysis and its accompaniments in various cases must depend on the nature of the original lesion or defect, as also the improvement or recovery seen in some instances. Some cases waste extremely, others but little or not at all, and it is conceivable that in some of these anomalous cases we have to do with spinal infantile paralysis of a chronic form. The electrical test, which would be of great value here, is often quite impracticable, and always difficult of application and interpretation in young children. It is possible in cases which show improvement that there is slight inflammatory or hæmorrhagic effusion in the meninges, syphilitic change in the arteries leading to softening, retrocedent tubercular growth, or even late compensation for previously arrested cerebral development. One very marked case I saw in a boy aged fourteen months, who for six months had had rigid spasm of all extremities, strabismus, retracted head, and frequent convulsions. After many weeks in bed, with excessive wasting, he gradually improved, and after six months seemed well in all respects. I saw him again after ten months more, when careful examination failed to detect any abnormality of mind or body. In another case of an undoubtedly syphilitic infant the symptoms apparently began at the age of six months. After about five months in hospital the limbs recovered and the child seemed well, but there was marked mental deficiency. Sometimes the paralysis is accompanied by more or less rhythmical movements. I saw one marked example of this in a girl of six years old, with a good family history, where the affection had been first noticed a short time after birth. She had "athetosis" without rigidity of arms, and rigid extension of legs. Another case with athetosis of arms and complete inability from birth to maintain equilibrium, though with no rigidity nor other symptoms, may be referred, I think, with many of the spastic cases proper, to some wide-spread defect of cerebral development. Occasionally I have seen cases with all the symptoms of the disease known as "multiple" or "insular" sclerosis which

may undoubtedly occur in early childhood, but post-mortem examinations showing the disseminated lesions in brain and cord connected with these symptoms in adults are not numerous in young children. Many cases however have been published under this title, some showing the "typical" symptoms, others with difficulty distinguishable from cerebellar or other tumours. At any rate the subject of definite and demonstrated "multiple sclerosis" has no special claim, in my opinion, to be discussed at length among diseases of childhood.

It is probable that in children these chronic spastic affections, from their multiform associations and differences of extent and degree, have a multiform causation; but it seems certain that in all cases they are due to deficiency, destruction or pressure in some part of the motor tract in the brain, as is evidenced also by numerous instances of brain tumour, especially of the pons, or of the cerebellum pressing on the pons, with spastic paralysis of limbs. In such cases however as are unaccompanied by any evidence of localised brain disease, of microcephaly, or of mental deficiency, ætiological diagnosis is very obscure.

In spite of several anomalous cases of tremors with movement without rigid spasm, which I have often thought are probably slight examples of the somewhat heterogeneous class of affections we have been considering, as well as of a few others with marked and wide-spread rigidity, and of the improvement of some of the numerous congenital cases usually referred to under the title of "spastic paraplegia," the *prognosis* on the whole must be very grave, especially when the affection is of long standing. It is very rarely that this set of symptoms, dating from infancy, is seen in adults, the patients tending to die young from various causes.

It need scarcely be said that *treatment*, in consequence, must be almost vain. We may try antisyphilitic remedies in all doubtful cases, but with scarcely any hope of success. In those cases which approximate in appearance, however much they differ pathologically, to the spastic paralysis of adults, but are however not progressive and may last indefinitely, exercise should be enjoined, and repeated passive flexion of the limbs may be of some slight use.

Chronic Paralysis with Atrophy of Muscles.

Under this very general heading I include certain groups of cases of marked weakness with wasting of muscles which begin almost always in childhood or early youth and are characterised by insidious onset and slow progress. In some with apparently stationary periods of considerable duration there is a tendency to death before adult age from general wearing out or from some intercurrent disease; in others there is sometimes comparative or complete arrest. The best marked group with fairly distinctive clinical characters is known as Pseudo-hypertrophic (or Duchenne's) paralysis; another, smaller and less clearly definable, may be classed generically as progressive amyotrophy, and diversely specified as differing from the well-known type-form which begins as a rule in the small muscles of the hand. Of this I shall allude to two species, known respectively as the "peroneal" and "juvenile" forms. This grouping is however exclusively clinical and confessedly imperfect, and by no means implies any conclusion or theory as to the pathology of these affections; for although there may be reasons for regarding some as due to primary disease of the muscles and others to disease of peripheral nerves or cord, it can scarcely be said that an exact pathological ætiology has been established in any, and I shall not enter here into any discussion of the differential diagnosis of myelopathies, neuropathies, or myopathies. In most of the probably heterogeneous class of affections there is usually believed to be a strong tendency to some kind of hereditary transmission.

Pseudo-Hypertrophic Paralysis.

This affection has long been recognised from its marked clinical characters which are fully described in the text-books and in Gowers' almost exhaustive monograph. It begins nearly exclusively in childhood, showing itself at first by a weakness of the lower extremities which causes the child to stand with his back arched inwards, to straddle and sway in walking, to fall down readily, and to rise from the floor and mount stairs with difficulty; later on, by apparent enlargement of certain muscles and wasting away of others with a tendency to "pes equinus" from

contraction of the calf muscles or weakness of their opponents and marked aggravation of all the symptoms; and, finally, by inability to walk or stand with extended wasting of muscles including those which were previously enlarged. The paralysis is apparently proportionate to the wasting and is not necessarily most marked in those muscles which are the seat of enlargement. The general health is usually good and the symptoms may remain stationary for long, but the disease once clearly established is perhaps always progressive and very few cases live to adult age. Death usually results from exhaustion or intercurrent disease, but our knowledge on this point is scanty as most cases are lost sight of by their recorders long before the end and from loss of their characteristic symptoms are probably unrecognisable by those in whose charge they die. One of my own well-marked cases, dismissed in an apparently stationary condition, was re-admitted under a colleague more than two years afterwards without a trace of muscular enlargement and quite incapable of any movement from excessive wasting of muscles, with universal rigidity, which suggested disease of the antero-lateral columns of the cord. Without the previous history the nature of this case would entirely have escaped notice. The boy died from chest mischief but a post-mortem was not permitted.

This disease may be suspected in the early stage from the straddling gait, the tendency to fall, and a certain difficulty in rising from the ground, the characteristic manœuvre of the patient's climbing up his own legs to assist the weakened extensors of the knee, hip, and spine being often postponed even until after some pseudo-hypertrophy has shown itself. In this early stage a marked hardness of the muscles which may afterwards become enlarged, and especially of those of the calves and buttocks, may sometimes be observed, and is an aid to diagnosis. But I must remark here, as bearing on the question whether the affection be ever recovered from or arrested in an early stage, that I have seen several cases, some of them after measles or other febrile diseases, where symptoms such as these, irresistibly reminding one of pseudo-hypertrophic paralysis, have lasted for weeks and completely disappeared, and others which have neither progressed nor shown any enlargement of muscles. I have seen, besides, a boy of six years old who had been slow and inactive for six weeks and unable to run for one week. There was marked

"lordosis," a straddling gait, inability to rise from the ground without touching the knees, and the calves and buttocks were noticeably large. The skin of the legs was mottled and the knee-jerk was absent on the right and very slight on the left side. After six weeks, during which time I had not seen him, all these symptoms were much less with the exception that both knee-jerks were now absent. A fortnight later he was quite well. I saw him finally six weeks after this when he was still without symptoms; the knee-jerks were both present and he walked and ran with ease.

In the established disease almost all the muscles of the body including sometimes those of the face may be more or less enlarged, but as a rule the most striking in this respect are the calf and buttock muscles, the spinati, and the deltoids. The pectorals, latissimi, and other muscles of the upper part of the body often waste both early and rapidly. The skin is most often mottled over the affected parts, especially after prolonged exposure. It has been often stated that the surface temperature of the enlarged parts is higher than that of the rest of the body; but I am convinced, from repeated and careful observations in four well-marked cases, that this is a mistake. The simple precaution of eliminating the disturbing element of different periods of exposure by altering the order of observations sufficiently establishes the error of the prevalent statement on this point. The knee-jerks are said to be lost only in the later stages. I found them present in one case of seven months' standing, the boy having been quite active till six and a half years old; absent in a boy of nine who had had difficulty in walking from the first when he was fifteen months old; and present, though slight, in a boy of ten who had been quite well till three years old. Faradic reaction in the nerve trunks is normal till very late, and then only diminished, owing, probably, to degeneration of the nerves; in the muscles it is always lessened from the resistance caused by the morbid fibrosis. Galvanism to the nerves does not show much change till late, but to the muscles produces a reaction diminished in proportion to the wasting of muscular fibre, and there may be a slight degree of the reaction of degeneration. In two of my cases I found that the faradic reactions in affected parts were altogether normal, one being of seven months, the other of seven years' duration, while in two more of several years' standing

there was diminution of reaction to faradism both in the nerves and muscles of the legs and scarcely any difference between the anodal and cathodal closure contractions with the galvanic current.

All my cases, including several doubtful ones and the probable instance of recovery, were boys, and in none was there any ascertainable trace of heredity direct or collateral. In many reported cases however it has been found that the disease occurs frequently in the same family, being apparently transmitted through unaffected mothers to their sons. Mental deficiency has been often noted. In one of my cases some obtuseness was reported from school, though the boy appeared quite intelligent in hospital; and in another speaking was delayed till the age of five. In the uncomplicated disease no sensory abnormality nor loss of control over bladder or rectum finds place. The clinical disease is certainly not congenital of necessity, but that there may be a latent congenital tendency is a hypothesis which cannot be disproved. In most cases no exciting cause can be found, and I regard as merely accidental the apparent development of one of my cases, aged seven years, out of a definite attack following on a fit and fever of infantile paralysis of the left and slightly of the right leg five years previously. The right leg recovered completely from the paralytic attack, and the left to a great extent, but on admission there was marked lessening in length and bulk in the whole left lower extremity where the surface temperature was persistently about 5 degrees lower than over the rest of the body. In this case there was marked wasting of the pectorals and deltoids, though the "spinatus" and "triceps" muscles were bulky, while the left calf as well as the right increased by $\frac{3}{4}$ inch in girth during six months' stay in hospital. Two years afterwards the boy could not stand; his deltoids were powerless and his buttocks wasted, but the triceps muscles and the calves were still very large.

The great weight of recent opinion about the pathology of this disease is on the side of a primary myopathy, although in some cases more or less wasting of the ganglionic cells in the anterior cornua of the cord and other morbid appearances have been found. In a case brought by Dr. Handford before the Pathological Society in 1889 there were the usual muscular changes, with degeneration of the diaphragm and heart muscle, and marked degeneration and atrophy of the peripheral nerves of the affected parts, which,

considering the absence of sensory symptoms, probably affected the motor nerve fibres only and was secondary to the muscular degeneration. The cord was mainly healthy, though there was occasional slight degeneration of the ganglionic cells in the anterior cornua, and an area of softening in the lumbar enlargement apparently due to hæmorrhage.

The muscular changes consist in great increase of connective tissue between both the bundles and the fibres themselves. There may be also an early overgrowth of fat. As the disease progresses much fat is deposited in the newly-formed connective tissue, and the muscular fibres are separated, atrophy, and disappear. Finally, all muscular structure may be obliterated, and the newly-formed fibrous tissue wastes, leaving nothing but fat and connective tissue and the fat itself may at last disappear. The morbid process is essentially the same in the muscles which waste from the first, and in those which have a stage of enlargement, and the pseudo-hypertrophy is mainly due to fibrosis.

The *diagnosis* of this disease is generally easy except at the beginning. It may occasionally be confounded with some of the rarer forms of progressive atrophy in children, presently to be noticed, and as I have often observed, and as Ross points out, with various cases of retarded development and want of co-ordination with or without signs of cerebral disease. I would especially remark here that the generalised forms of paralysis not seldom seen after diphtheria and probably some other diseases give rise to some of the symptoms which many thoughtlessly regard as pathognomonic of pseudo-hypertrophy. I have more than once seen the action of climbing up the legs as well as the waddling gait in clear cases of diphtheritic paralysis which perfectly recovered, where doubtless the same muscles were weakened as in the disease we are considering. A doubtful diagnosis may possibly be cleared up by extracting a small piece of muscle with one of the instruments invented for the purpose and examining it microscopically, but even when repeated, as it must always be, such a procedure is generally unsatisfactory in its result and not without risk, nor is it a worthy cause for administering an anæsthetic without which the operation is inadmissible.

The only hope for treatment, ignorant as we are of the cause of this disease, lies in the possibility of aiding any natural tendency there may be to arrest or recovery. I have seen cases improve

considerably in hospital with good food and tonics, but one of these at least made subsequently steady progress downwards. Duchenne says that he arrested two early cases by faradism, but in the light of the case quoted above of recovery under ordinary tonic treatment from symptoms which were at least very suggestive if not identical with those of the recognised disease such a statement is of little value. I would recommend in all early and all doubtful cases the best hygienic conditions, regulated exercise, shampooing of the affected limbs, and arsenic, iron, cod-liver oil, or a combination of these, as possibly helpful towards improvement or cure.

The Peroneal Type of Progressive Amyotrophy.

This affection has been clinically isolated of late years, and cases in point have been described by Leyden, Ormerod, Charcot and Marie, Tooth, Herringham, and others. I showed at the June meeting of the Neurological Society in 1890 three cases in one family, recording them in "Brain" (vol. xiii. p. 456), and would refer the reader especially to Dr. Tooth's papers in the same periodical (vol. x. p. 243), and in the St. Bartholomew's Hospital Reports (vol. xxv.) for a full account of this class of cases. In established cases there is wasting and weakness of the legs often with markedly diminished sensibility, and the feet are in the "varus" position. At least in the later stages there is absence of reaction both to faradism and galvanism. Subsequently to the leg affection there is wasting with or without similar anæsthesia and loss of electric contractility in the small muscles of the hands which ultimately assume the claw-like appearance of ordinary progressive muscular atrophy. In some early cases there are pains with tremors and unaltered electrical reactions and in a subsequent stage there is the reaction of degeneration. Heredity, though often marked, is not always present. Dr. Ormerod's cases and my own were both groups of three, consisting of father son and daughter; and the symptoms in all save the father in the latter group set in immediately after an attack of measles. The disease seems always to begin in childhood or early youth and probably affects first the small muscles of the foot. Muscles besides those already mentioned may subsequently suffer, but there seems to be no doubt that many cases are arrested for at

least an indefinite time after the legs and forearms are affected. In all my own cases the muscles of nearly all the rest of the body were normal in every respect, the girl, aged 17, affected since the age of seven, having become no worse for a long time, and the father, aged 42, first affected at 17, having been in a stationary condition for many years. The gait in all these cases was high-stepping (not spastic, as at first sight appeared) owing mainly to foot-drop from peroneal weakness, and the knee-jerks were present with no ankle-clonus.

The pathology of this affection is not certainly known. On the whole, considering the at least occasional presence of fibrillary tremors in the early stages, the occurrence of pain in some cases, and the anæsthesia in many, a primary peripheral neuropathy seems perhaps to be indicated. Some regard the lesion as situated in the cord, and others as starting in the muscles. Too few necropsies have been published for the establishment of any point of ætiological importance. The immediate antecedence of measles in the cases above alluded to is to be remarked in connection with other forms of paralysis apparently excited in the same way, but it can only be regarded as a favouring condition for the neuro-muscular break-down which constitutes the disease. There is a general clinical likeness between this affection and pseudo-hypertrophic paralysis, but the pathology is even still more doubtful, and nothing of value can be said on the matter of treatment.

The "Juvenile" Type of Amyotrophy.

Of this form, described by Erb, I shall say but little, having only seen cases observed and published by others. Dr. Savill showed some very good examples at the Neurological Society in June, 1890. The symptoms of wasting and weakness attack first and are often long limited to the shoulder and upper arm and the buttocks and thighs; there is lessening or loss of the knee-jerks, no fibrillary tremor, and no reaction of degeneration but only diminished electric contractility. The disease is said to begin in later childhood or early youth. In some cases wasting of the face and tongue has been observed. As with the previously described cases there is nothing as yet approaching to certain ætiological knowledge of this affection, and it has only clinical claims to a separate position.

ON ISCHÆMIA AS A CAUSE OF PAIN, RIGIDITY, AND PARALYSIS, AND A WARNING AGAINST GANGRENE.

By W. G. SPENCER, M.S.

Attention to the premonitory symptoms of gangrene is of the greatest importance, because that may be the means of warding off the evil. The term ischæmia, first used by Virchow,* is employed to indicate the partial diminution of the blood-supply to any part. This partial diminution of the circulation may give rise to a special form of rigidity and to paralysis, and is especially seen in the limbs, particularly the lower ones. The obstruction causing the rigidity and paralysis occurs under various forms, and a number of experiments have been performed on animals which throw light upon the pathology. I will first record some of the clinical conditions, and leave the experiments until later in the paper.

This condition was well marked in some cases of ruptured popliteal aneurism which have recently been in the Westminster Hospital.

A man, R., aged 40, who had drunk heavily, was admitted under my care in August, 1889, with a double popliteal aneurism. The one on the left side was apparently thin-walled and some two inches in diameter. On the right side a hard swelling occupied the popliteal space, in which pulsation was not well marked. On the previous day when stepping from an omnibus he had felt something give way in the right popliteal space, which was followed by a rapid swelling and a partial inability to move the limb. Before this accident the aneurism on the right side had been, according to the patient, the same in size as that on the left. The right leg and foot were slightly œdematous, and it

* Virchow, Handb. des Spec. Path. u. Therap., 1854, Bd. i p. 122.

was doubtful whether any pulsation could be felt in the arteries about the ankle, but the toes were quite warm. During the first night after admission he was very restless, and an attack of delirium tremens began. The next morning the diffusion of blood from the ruptured aneurism had evidently increased, consequently on that day, August 7th, I ligatured the superficial femoral artery at the apex of Scarpa's triangle. This was done in preference to cutting down directly upon the aneurism in the popliteal space, which appeared very hazardous in face of the condition of the patient. On August 8th the foot was warm, and the effusion had ceased to extend. The patient was very excited during several succeeding days and nights, and in spite of the confinement of the limb upon a splint and the restraint by an attendant, he knocked the limb a good deal against the bed. In consequence the swelling in the popliteal space became inflamed and increased considerably in size, and concurrently with the increase of swelling the foot became pale, cold, and then blue as far as the bases of the metatarsal bones. There was severe pain, especially in the heel, the muscles of the calf were hard and tender, and he lost the power of moving the foot. The blueness of the toes continued until August 16th in about the same condition. Meanwhile the inflammation in the popliteal space had become less, and the aneurism could then be felt as a hard mass. Cotemporaneous with this reduction of inflammation the circulation returned in the foot, the anastomatic circulation having been greatly impeded by the inflamed state of the popliteal space. The circulation became normal up to the bases of the metatarsal bones, and also extended to the blue area in front as far as the toes, but in this blue area the capillaries had evidently undergone degeneration in consequence of the absence of circulation, so that when the circulation returned a red suffusion of the blue area took place, the blood being extravasated from the capillaries. The front part of the foot beyond the bases of the metatarsal bones afterwards underwent dry gangrene. The pain in the heel and the rigidity and tenderness of the calf muscles continued, and later on the rigidity passed over to contracture, leaving the muscles shrunken and hard, and the foot fixed in extension. The wound made for the ligature of the femoral artery healed by first intention, and the patient remained in bed for four months in the hope that an amputation

across the middle of the foot might be done. But during his delirium he injured the outer side of his left leg by making three bruises over the fibula. These bruises later on broke down into abscesses and the fibula became exposed. The exposed fibula was only slowly covered again by granulations, and after lying in bed four months the man was becoming exhausted. On December 11th I amputated the leg at the knee-joint. On examination of the leg it was found that the muscles had undergone changes of acute inflammation, the muscle fibres had degenerated, and their place had been taken by inflammatory fibrous tissue. Mr. De Renzi kindly made some sections of the altered muscle.

I also saw another case in which similar rigidity and paralysis occurred in a man aged 27, under Dr. Hebb, suffering from the results of acute endocarditis, amongst which was a popliteal aneurism which had become diffused. With the diffusion there occurred severe pain of a cramp-like character in the calf and heel, and loss of power in moving the calf muscles, which could be felt to be rigid. Compression of the femoral artery was tried, and this relieved the pain and lessened the rigidity of the calf; but the general anasarca rendered considerable force necessary in the application of the pressure in order to control the artery. Consequently the pressure had to be relaxed to allow the patient some sleep. The relaxation of the compression was followed by a return of the pain and rigidity. The patient died the next day. At the post-mortem it was found that the blood had been diffused from the ruptured artery, without the formation of a sac. The muscles had not undergone any change.

In a third case, under Mr. Macnamara, of a ruptured popliteal aneurism, there was a similar rigidity and paralysis of the calf muscles, but moist gangrene rapidly set in, so that the muscles were destroyed by the gangrene before an examination could take place.

In less severe cases a slight pain or paresis may ensue from ligature of the large artery of the leg. In a case lately under Mr. Macnamara, in which he ligatured the external iliac for an aneurism in Scarpa's triangle, the patient, after recovery, and being able to walk more than a mile, complained that in walking fast he was liable to cramp in the calf and temporary loss of power.

In a case I saw, whilst a student, the leg remained extremely weak after ligature of the femoral for popliteal aneurism.

Broca* describes as a result sometimes following ligature of the main artery of a limb, disturbances of sensation, numbness, cold, or more or less pain; and in other cases, paralysis and atrophy.

Porta† collected 14 cases in which atrophy and paralysis occurred as a direct consequence of the division of an artery, excluding any nerve lesion. Thus, in a case of a man aged 32, noted by Porta, the ligature of the brachial artery for aneurism was followed by formications, cramps, weight, rigidity, and inertia. At a later stage the temperature and pulse in the limb were regained. After the patient's death, which took place from bilateral pleurisy, the ligature was found to have included the artery and one of the venæ comites, but no nerve.

It is, however, in cases of partial or complete obstruction of the lower end of the aorta and iliacs that ischæmic symptoms are mostly seen. I may first refer to an affection of horses, described by Goubaux,‡ Boullay,§ and others, under the term of intermittent lameness (*Claudication intermittente*) due to an obstruction in the abdominal aorta or iliacs. In a well-marked case the animal when at rest, or walking slowly, might show no signs of disease, but when made to trot or pull a heavy load, it would, within five minutes, begin to go lame, and then be seized with a sudden rigidity of the hind limbs, so that there was inability to move them. The animal then fell, sweating profusely and respiring rapidly, indicating severe pain, whilst the hind limbs were rigidly extended. After resting on the ground 15 or 20 minutes, the animal could then get up and walk without lameness. The same symptoms recurred on any extra exertion. The pathological lesion proved in many cases to be a rupture of the internal coats of the aorta near its bifurcation from overstrain, and in all cases a thrombosis more or less extensive of the end of the aorta, iliacs, and fermorals. In some cases only one limb was affected. Symptoms of a similar nature occur in man. One of the first cases described, although not accompanied

* Broca, Des Anévrysmes, 1856, p. 501.

† Porta, Delle Alterazione patologich delle Arterie per la legatura e la torsione, Milano, 1845, enfol 386—391.

‡ Goubaux, Reçueil de Médécine Vétérinaire pratique, 1846, T. iii. p. 578.

§ Boullay, Archive gén. de méd., T. xxviii., 1831, p. 425.

by a post-mortem examination, is that related by Gull,* entitled "A case of Paraplegia from Obstruction of the Abdominal Aorta." A man whilst at work, and stooping, felt a sudden pain round the loins, and a desire to defæcate; the pain went off in a few minutes, but when he returned to work the pain came on again, and soon there was entire loss of sensation and motion from the loins downwards, together with paralysis of the sphincters. After a few days sensation returned, and he was able to take a few unsteady steps, but the attempt to use his legs soon brought on an increasing numbness in them. The patient was in Guy's Hospital in June, 1855, when a thrombosis of the abdominal aorta and its larger branches was diagnosed, both from the absence of pulsation at the end of the aorta, iliacs, and femorals, and also from the marked dilatation of the anastomatic circulation. The patient suffered from weakness and numbness in the legs, the muscles being thin and the gait languid.

Charcot,† after describing the "Claudication intermittente" as it occurred in horses, mentions the case of a man aged 54, in whom the right iliac had become transformed into a partially obliterated aneurism.

Jean‡ recorded the case of a woman with complete obstruction of the aorta, from the diaphragm downwards to the ends of the iliacs, the femorals being patent. For some time before her death, she was accustomed to suffer from complete paraplegia on any extra exertion. After an attack she would keep her bed several days, and was then able to walk again as usual, but with the least signs of fatigue her legs began to drag afresh, and she was obliged to rest. She continued like this for two years, sometimes being able to get about and do her work, at other times it was impossible to take any exercise. Paraplegia increased before her death, which occurred from enteritis. The rest of the aorta was normal, whilst the abdominal aorta was completely obliterated, as were also the external and internal iliacs.

Ollivier§ saw a case of a man aged 42, which commenced with severe pain as of cold in the right foot; it was taken at first to

* Gull, Guy's Hospital Reports, 1857, 3rd ser., iii. p. 311.

† Charcot, Gaz. Méd. de Paris, 1859, p. 282.

‡ Jean, Bull. de la Soc. Anat., Paris, 1875, 3me ser., p. 282.

§ Ollivier, Observation pour servir à l'histoire de la Claudication intermittente chez l'homme.

be chronic rheumatism. When resting he felt no pain, but whenever he tried to return to his work he could hardly walk for two or three minutes without severe pain in the ankle or calf. In cold weather the limb became icy and painful, whilst during warm weather the pain was nil, or could easily be borne. He gradually lost power in the limb, and the muscles atrophied. He could walk for seven or eight minutes without suffering, then would suddenly stop, being seized with violent pain in the right calf and cramps in the foot. No arteries could be felt beating in the right leg, although the femoral was sought for with the greatest care.

In a case by Terillon,* a man aged 27, who had drank much, began to suffer from sensations of cold in the feet and legs. In July, 1885, pains in the left foot appeared, and soon after walking was rendered painful by the occurrence of a sharp pain in the calf which obliged him to stop. The pains increased, whilst the legs still retained their normal appearance. After a time the pains involved the whole leg, and then all movement became impossible, whilst the patient continued to have a burning pain in the legs. On September 25th gangrene commenced in one leg, which had to be arrested by amputation. After the amputation pain disappeared from the thigh which formed the stump.

Another set of cases are those due to compression of the arteries supplying muscle by tight bandages, such as were first noted by Volkmann,† and fully described by Leser.‡

Volkmann saw a case in which a plaster of Paris bandage had been applied to the forearm immediately after the occurrence of fracture without any intervening wool or soft bandage, and the plaster allowed to remain on in spite of the swelling of the hand. The first result of such a tight bandage was a venous congestion and swelling of the hand, followed by acute inflammation of the muscles of the forearm. The muscles were intensely painful and rigid, with inability to move the fingers; whilst attempts at passive movement increased the pain to agony. If the plaster were removed soon after the commencement of the inflammation the muscles lost their rigidity, and movement was gradually regained; but in the cases where the inflammation of the muscles

* Terillon, Revue de Chirurgie, 1886, vi. 813.

† Volkmann, Ischaemische Muskellahmungen u. Kontrakturen, Centralblatt für Chirurgie, 1881, s. 801.

‡ Leser, Volkmann's Klinische Sammlung, No. 249 (Chirurgie, No. 77).

had reached to an extensive degree, the muscle fibre degenerated, and its place was taken by fibrous tissue. Contractures involving the fingers and forearm were thus produced against which after-treatment was useless. With the muscular degeneration there occurred the usual alteration in the electrical reactions.

A bandage a degree tighter applied to a fracture of the forearm produced complete arrest of the blood supply, and consequently gangrene.

There are various other ways in which the ischæmic muscular paralysis may arise. Such is the case described by Petersen* of a girl 4½ years, in whom the brachial artery was occluded by a compound fracture of the humerus, in consequence of which there was loss of movement in the muscles of the forearm, which became changed into hard fibrous tissue, and the strongest faradic current could be borne without pain.

Mollitor† has described a case in which amputation of the arm was done ten days after a compound dislocation at the elbow, complicated by a tearing of the brachial artery. He describes the early changes of degeneration in the muscular fibres, which leave only a few of them persistent, and it is only these few therefore that are capable of forming new fibres.

The foregoing cases may be taken as the more severe forms which attend the partial cutting off of the blood supply, and therefore they are the stage immediately preceding gangrene. But there are other slighter conditions in which no doubt gangrene is not so imminent, but which are of more common occurrence.

We may first notice the partial loss of power and the stiffness in the muscles due to severe cold, in which the blood supply is only partly impeded. Cold of a more severe degree causes gangrene (or frostbite). Again, there is the partial loss of power and rigidity of the muscles which attend their over-use, such, for instance, as the calf muscles after over-exertion, in which the muscles are hard and painful, and have partially lost their power. In this case the blood supply is insufficient to meet the increased wants of the muscle.

A very important form is that of cramp occurring in swimming, and which frequently causes the loss of life to even expert

* Petersen, Archiv für Klinische Chirurgie, 1888, xxxvii. 675.

† Mollitor, Beitr. zur Klin. Chir., Bd. v. Heft 2.

swimmers. We have in this condition the effect of cold upon the actual circulation in the limb, the demand of the muscles of the limb for more blood to meet the waste produced by the exertion, together with the general lowering of the heart's action, by the combined action of cold and fatigue. The loss of power and the rigidity of the muscles come on rapidly, as soon as the circulation of the limb is reduced below a certain degree, by a combination of the above causes.

But besides local causes producing local results, we have local conditions attending general disease. Nothing is more common in cases of general anæmia than slight tinglings and creepings, or even aching pain, in one or other of the extremities, together with loss of muscular force. Such are some of the early symptoms, for instance, of Reynaud's disease, in which the part which has been the seat of tinglings or pain and loss of power may later on suffer from gangrene.

Intense pain may occur on one side of the scalp in cases of hemicrania, as described by Du Bois Reymond* and Brunner†, from spasm of the arteries of the side of the face.

The same series of symptoms are of great importance in old people, in whom a narrowing of the smaller arteries by calcareous disease produces a tendency to senile gangrene. Such are tingling, creeping, numbness, aching pain, and loss of muscular power in the part. In these cases there may be also diminution of the surface temperature, and the pulse, for instance, about the ankle may be lessened in volume.

Mr. Hutchinson‡ has lately recorded two such cases under the title of "arterial anæsthesia."

Ischæmia affects also the brain, and is one of the premonitory symptoms of apoplexy. Many suffer from pain or temporary paresis who later on undergo an attack of apoplexy. There is in such cases a narrowing of the vessels by the stenosis produced by the atheroma.

I may refer to a paper by myself and Mr. Horsley,§ which showed that the blood supply could be cut off from the middle cerebral artery for a short time by compression of the carotid

* Du Bois Reymond, Zur Kenntniss der Hemikrania, Du Bois Reymond's Archiv, 1860, s. 461.

† Brunner, Petersburg Zeitschrift, 1871, s. 260.

‡ Hutchinson, Archives of Surgery, 1890, vol. i., No. 4, April, p. 339.

§ Spencer and Horsley, British Medical Journal, 1889, vol. i.

in monkeys, which resulted in an anæmia of the cortex and a loss of electric reaction in the motor area.

There are many experiments recorded, which help to illustrate the pathology of the conditions which have been mentioned above. The earliest were made in researches upon muscular movement.

Steno* is said to have originated the experiment which goes by his name, viz., the ligation of the abdominal aorta. Having tied that vessel in a fish, the animal ceased to move its tail.

Willis,† in describing his experiments, says, "*Observavit ille quod in cane vivo, ligata aorta descendente, sine prævia sectione posteriorum omnium motus voluntarius cessaret quoties vinculum stringebat, iterumque tot vicibus rediret quoties nodum laxaret.*"

The experiment of ligature of the abdominal aorta has been repeated many times, especially in rabbits, the result being in that animal, first a gradual loss of sensation and motion in the hind limbs, followed later on by rigidity.

Brown-Séquard‡ showed that this rigidity was produced in the muscle by the same process that occurs in cadaveric rigidity. The rigidity passed off if the circulation was soon allowed to return; but if the constriction was maintained, the rigidity remained until the death of the animal. The compression or ligature of the abdominal aorta in the rabbit, cuts off the blood supply to the lower limbs, which is not compensated for by a sufficient anastomosis between the internal mammary and lumbar arteries on the one hand, and the epigastric and circumflex iliac on the other. The anastomosis is peculiarly small in this species, and the manner of experimentation by making an incision through the loin, and by cutting through the lumbar vessels, diminished further this anastomosis. But it is incorrect to assume that the obstruction or ligature of the abdominal aorta in other animals is a cause of paraplegia. In the rabbit, if the ligature be applied in front of the hinder end of the spinal cord, there is a complication produced by diminishing the blood supply to the spinal cord itself, through the lumbar vessels on the peripheral side of the ligature.

* Steno v. Haller, Elementa Physiologiæ, Lausanne, 1762, Tom IV., Lib. XI., Sect. xix., p. 544.

† Willis v. Longet, Traité de Physiologie, T. I., 2me partie, p. 34.

‡ Brown-Séquard, Leçons sur les principales formes de Paralyse des membres inférieures, 1865, p. 68.

Sir Astley Cooper* showed that if the abdominal aorta were ligatured immediately above its bifurcation, by a median incision through the abdominal wall, with ordinary surgical care, the ligature did not produce paralysis of the hind limbs, and I was able to show, by experiments on monkeys, that the abdominal aorta might be ligatured below the origin of the renal arteries, by employing all antiseptic precautions, without any ill effect to the animal. This certainly applies to man, because there are many cases described of congenital or acquired occlusion of the abdominal and common iliacs without the production of any symptoms, and the cases in which the abdominal aorta has been ligatured in man, although done as a last resource against aneurism which had already burst, yet as far as they go show that the circulation was not cut off from the legs by the ligation.

The compression of the abdominal aorta has also been employed in the cure of abdominal aneurism, and also, for instance, for the control of uterine hæmorrhages, and in these cases the collateral blood supply to the limbs has been sufficient.

In order to produce the ischæmic symptoms of pain, paralysis, and rigidity already referred to, it is necessary for not only the lower end of the abdominal aorta, but also the common iliacs and the external iliacs, to become obstructed, so that the collateral circulation reaching the femoral through the epigastric and circumflex iliac is cut off; whilst, in order to produce actual gangrene, it is generally necessary that the thrombosis should occlude partly or wholly the femoral artery also, as far as the bifurcation of the popliteal.

Lallement† saw a woman, aged 42, who died of cancer of the stomach, in whom the abdominal aorta was completely occluded at the level of the cœliac axis, and the vessels below as well as the common and external iliacs were obliterated down to the crural ring. At no time during the disease was there any trouble in motion or sensation in the lower limbs.

In Munro's‡ case the aorta was completely obliterated at the lower end by an aneurism, but there was no appearance of weakness, numbness, or paralysis in the lower limbs. It is the condition, therefore, of the femoral artery which is of critical

* Astley Cooper, Med. Chir. Trans., vol. ii., p. 258.

† Lallement, Gaz. Hebd. de Méd. et de Chirurg., 1866, 2me ser., T. iii. p. 71.

‡ Munro, Med. Chir. Review, 1827, vol. i. p. 481.

importance. As long as it is patent the recorded cases show that gangrene of the extremity is not likely to occur unless the force of the circulation is much reduced by disease elsewhere.

But when the femoral artery becomes changed into a hard cord by thrombosis, gangrene will appear almost certainly within a few days.

The treatment of the condition included under ischæmia follows on the recognition that the cause is the partial diminution of the blood supply, and that there is a danger of the diminution going further.

Attention is chiefly required to the protection of the affected part from cold. The application of warmth to dilate the vessels and massage is beneficial, as is experienced by everyone who suffers from muscular fatigue. In old people, when the cause is due to sclerosis of the vessels, and in conditions of general anæmia, tonics have the effect of increasing the general circulation. Of drugs, opium is generally the most serviceable in improving the circulation of the extremities in old people, apparently by dilating the capillaries.

ON SOME CASES OF ANÆMIA, AND THE "MOBILE BODIES" IN THE BLOOD.

By Dr. HEBB and Dr. WILLS.

The following cases are intended to illustrate a few of the phases of anæmia, and to point out the existence of a particle of protoplasm, endowed with motion, which has been found in the blood.

Though the number of cases cited is limited, and the number of microscopical observations may appear small, yet these have been really far more numerous than the written statement takes notice of, and the paper is to be regarded chiefly in the light of "protecting the authors' rights" should further investigation demonstrate the constant presence of the mobile body under certain circumstances, and its absence under others. For example, assuming a pathogenic property for the zooid, we should not, *cæteris paribus*, expect to find it in cases of anæmia due to loss of blood, and so on.

The case first quoted would be known, if the blood were the diagnostic criterion, as pernicious anæmia; the second as hæmorrhagic purpura; and the third as chlorosis.

Charlotte P—, æt. 50, a cook, was admitted under Dr. Sturges, on February 10th, 1891, complaining of general weakness, headache, pains across the back, and loss of sensation in the legs. She knew nothing of her family, owing to her being a foundling. She had suffered from no previous illness. She was a teetotaler, and for 33 years had worked very hard as a cook.

Three years before admission she reached the climacteric. Since then she had noticed that she had been losing her former good colour, and getting very pale and weak. A year later she began to faint, and this continued until admission. In one of the early fainting fits she fell down in the snow, and remained there for 10 minutes. On recovering consciousness she found

her legs were numb, and they remained so ever since. In 1889 she was some time in Beckenham Hospital with these symptoms, which, however, improved considerably during her stay; but during this period, in the course of three weeks, her hair, originally a golden brown, became quite white. Since then her breath has been getting very short, and at Christmas, 1890, she was obliged to give up her work.

She was, when admitted, exceedingly pale, with white hair and eyebrows, but her lips and tongue did not show a proportionate loss of colour. She was thin, but not markedly so. Temperature normal. Lungs normal. Heart's apex beat not felt. Præcordial dulness normal. Sounds feeble. First sound at apex prolonged, but no bruit. Urine 1014, albumen a trace. There was complete anæsthesia and analgesia of the legs from the knees downwards and on the left side. This extended about 3 inches above the knee. Her legs also were insensitive to hot applications.

Microscopical examination of blood March 12th, and three subsequent occasions. "The blood is a marked example of poikilocytosis, the red disks showing the most various forms. They are also very unequal in size, varying from 3 to 11 or even 12 μ. White corpuscles not apparently in excess. Under a magnification of about 600 ($\frac{1}{8}$ apochromatic) cylindrical mobile bodies were observed at each examination (4). These varied in length from about 3 to 5 μ. They usually showed an expansion at each end, and sometimes in the middle. Several possessed a double or furcate extremity. These bodies, in which no definite structure was observable, passed up and down the field, frequently with considerable agility, but for the most part their movement was slow. No pigment was observed in them, or any free pigment in the blood."

Examination of blood with hæmocytometer was not made in this case.

Her condition remained much the same up till March 15th, the time of her discharge. She regained colour slightly, and the albumen disappeared from her urine. She afterwards became an out-patient under one of us, and during the several months she has been from time to time under observation, her condition has not altered.

W. H., male, aged 19, servant at a shoeblacking institution,

was admitted under Dr. Sturges, April 8th, 1891, for bleeding from the gums.

Family history unimportant. Personal history: has had two previous attacks of bleeding from the gums, accompanied by spots about the body. States that he bleeds freely when he cuts his finger, or when a tooth is drawn.

The present attack, the third, came on suddenly on the morning of April 5th, with bleeding from the gums and sore throat. Next day spots appeared upon the skin of the body. Vomited twice, and the vomit contained blood. On April 8th fainted away while walking.

The condition, on admission, is described as being well nourished. The surface of the body is covered with hæmorrhagic spots, the mucosa of lips and gums being similarly affected. During his stay in hospital had several attacks of gencival hæmorrhage. Was discharged April 27th. Re-admitted October 21st. Had been quite well up to the 15th October, when he was seized with severe headache and pains in the limbs. On the 16th the gums began to bleed and hæmorrhagic spots appeared all over the body.

Is plump, but anæmic, and the cutaneous surface is covered with red or purple blotches of various size. Systolic bruit audible all over the precordia. Lungs nil. Urine nil. Microscopical examination of the blood, October 25th (when drawn from finger is thin and watery): red disks of very pale yellow hue. Are fairly equal in size, their average diameter being about 7·5 μ. Shape circular, there being almost no tendency to poikilocytosis.

Numerous "mobile bodies," sometimes two and even three, observed in the same field of microscope, the power used being $\frac{1}{6}$ apochromatic (= × 600 about). "The mobile bodies are for the most part cylindrical or cylindroidal in shape, with slightly bulbous extremities. They frequently exhibit lively movements, which are apparently independent and spontaneous. They move upwards and downwards, and to and fro across the field, and sometimes wander right round a corpuscle. The adjacent corpuscles do not move unless the 'body' impinge against them."

The foregoing description of the mobility of the bodies is not to be taken as indicating an enormous velocity, indeed, as a rule,

the contrary is almost the case. Yet a few have exhibited quite lively if not rapid movements.

Though most of them are cylindrical, some of these bodies are spindle-shaped and Y-shaped. Their average length is about two-thirds that of the diameter of a red disk. Their width is about or less than half that of a red corpuscle when seen on edge. They do not contain any pigment, nor is there any free pigment in the blood.

Examination on 29th showed that there were fewer bodies, but in other respects the blood seemed very much the same as on 25th. The patient's condition seemed much improved.

Examination of blood with hæmocytometer showed the red corpuscles to be 26·6 per cent. of normal, and the proper relative number of red and white cells.

November 2nd.—Examination of blood this morning showed a moderate number of mobile bodies, several exhibiting extremely lively movements.

November 3rd.—Complained of severe general pains, was seized with great breathlessness, and died after a few hours.

The post-mortem examination revealed only extreme pallor of the tissues, thin watery blood, and diffuse capillary hæmorrhagic extravasation almost exclusively confined to the sub-pleural and sub-pericardial regions.

Post-mortem microscopical examination of the blood showed one or two mobile bodies. In numerous red corpuscles linear forms resembling in size and appearance the mobile bodies were observed, but they were quite devoid of movement.

Kate H—, æt. 17, a scullery-maid, was admitted into Adelaide Ward under Dr. Sturges on October 21st, 1891, having been transferred from Mr. Macnamara's Ward, where she had been for four days.

She had been in service in Gloucester, in a large household, where she had been worked very hard and only got out for a few hours on alternate Sundays.

No family history known, except that her father died of a broken blood-vessel.

She had had infantile complaints but no other previous illness, and though always pale, was a strong, healthy girl up to one month before admission, when she began to suffer from headache, shortness of breath, sickness, and constipation, and she got

rapidly paler than before. Her catamenia, previously regular, ceased at the end of July. A week before admission she became so ill that she had to take to bed. When she came into hospital her temperature was 102°, she was extremely pale, lay with her eyes closed on account of the severity of her headache, which, at first limited to the right half of the vertex, had latterly spread to the left. Her pupils were equal, did not act very readily. No facial or other paralysis. Pulse 88. Apex beat diffused, in normal position, soft systolic mumur over pulmonary area, and not conducted into axilla. No other abnormal physical signs in chest or in abdomen. Tongue rather dry and furred in centre. Bowels constipated. On October 22nd her temperature fell to normal, and her headache, which had remained since admission so severe as almost to prevent her sleeping, became much better. She was sick on 22nd and 23rd after enemata, but she rapidly improved.

Microscopical examination of blood, Oct. 25, 1891: well-marked poikilocytosis, but megalocytes (large red disks) were not observed, the tendency of this blood being to smallness of size. Average diameter was 6 μ, that of leucocytes 7—8 μ. Of small red disks (microcytes) there is quite a large quantity.

"Mobile bodies" having similar characters to those previously described in the two preceding cases were observed, but only a small number.

Examination of blood by hæmocytometer showed the red corpuscles to be 68 per cent. of normal and no excess of leucocytes.

Besides these three cases, which are chiefly intended to be the peg upon which to hang a few remarks, numerous other cases representing different phases of anæmia have been examined. For example, to mention a few at this present time in hospital: (1) A girl, aged 17, admitted for anæmia and purpura. Blood when drawn thin and watery, corpuscles of average size, almost no tendency to poikilocytosis, only a few mobile bodies. (2) Two cases of simple anæmia in females, one aged 18, the other 23; in both these instances the red disks are smaller than normal, and in both the variations of shape are characteristic of anæmia, in both a few mobile bodies were observed. In the first of these cases the temperature is 102°. (3) Male child, aged 3½, very pallid, spleen very large, said to be subject of hereditary syphilis;

blood when drawn thin, pallid, and watery, no excess of white corpuscles, a few mobile bodies.

It will be seen that the foregoing cases illustrate different aspects of anæmia, but they all have one factor in common, the mobile body. In none has there been loss of blood from traumatism, ulceration, or the like. Two would be described as purpura hæmorrhagica, some as chlorosis. In some there is increased temperature. In some constipation. In none organic disease of the heart. In one greatly enlarged spleen, but without any increase of the white blood corpuscles.

With regard to the mobile bodies themselves, and which were first remarked in the case of Charlotte P—, it was at first concluded that they were merely one of the deviations so frequent in the red disks in anæmia, deviations of size and shape known as poikilocytosis. But the frequent appearance of one kind of body with an approximately definite shape and size, a body further characterised by specific movements to and fro, up and down, and circumnavigatory, their cylindrical shape, a shape perfectly well seen during the various gyrations of the body, rendered it almost probable that these bodies were independent of the red corpuscles or of the blood plates. Besides this they have also been noticed in cases of anæmia where the blood has given no evidence of poikilocytosis, and though they have been frequently sought for in normal blood, these mobile bodies have not as yet been detected.

That mobile organisms may exist in the blood of animals, and that without doing any particularly obvious harm to their host, may be gathered from the fact that flagellated organisms have been detected therein in the rat, &c. (*see* Crookshank, "Journal Royal Microscopical Society," 1886, p. 913, where reference is made to the parasite of the rat and of other animals), not to mention the Filaria sanguinis, the existence of which in about 10 per cent. of the inhabitants where this animalcule is endemic, seems to have been verified beyond dispute. But on the whole it is better to consider that the minute parasites of the animal as well as of the vegetable world are potentially noxious. For example, the surra parasite, a flagellated organism, is pathogenic to horses, and the plasmodia of malaria are now pretty well accepted as being the cause of this disease.

There is, therefore, no occasion for excluding the supposition

that mobile organisms may exist in human blood with or without a harmful or pathogenic action.

Yet even assuming that these bodies are imported organisms, it does not necessarily follow that they are related directly to the anæmia, that is, related as cause and effect.

If not parasites at all, then these bodies must be direct derivatives of red corpuscles, or, indeed, very small red disks, which have assumed a peculiar shape and become endowed with motion.

Such a supposition is, of course, quite possible, and in this case these pieces of protoplasm may represent a moribund condition of part of a corpuscle which has escaped from its proper quarters to an uncomfortable environment, for these bodies are best seen in a very thin layer of blood, and after it has been removed from the body a little while.

Still even if altered corpuscles or pieces of human plasma, the observation of their existence is interesting, for as far as we know mobile normal corpuscles have not been hitherto noticed, although bright, highly refracting monads, exhibiting brownian movements, are, if we do not mistake, referred to somewhere. At any rate we ourselves have frequently observed these bright monads in human blood.

The difficulty about making an observation on human blood, and afterwards correctly interpreting it, is much greater than may be supposed. For example, Hayem, who has spent a lifetime in observing human blood, was quite content to pooh-pooh the laverania of malaria, for, said he, anybody could see this sort of thing any day in anæmic blood. Yet the hæmatozoa of malaria are now almost universally accepted.

With regard to these mobile bodies it seems almost difficult to believe that they have not been seen before, but it is quite possible that they have been dismissed as corpuscles seen edgewise. But then these bodies are not more than $\frac{1}{2}$—$\frac{1}{3}$ the thickness of a red disk, and not nearly so long. And then they move.

It may further be admitted that there has been ill or only unsatisfactory results from staining films of blood, fixed with osmic acid vapour and dried on cover-glasses, for these show only blurred images of something which might have been, images which closely resemble most of the photomicrographs of pathological objects.

But it is far different with fresh blood. In this there is something to be seen; that something is a thin cylindrical moving piece of protoplasm. But to do this one requisite is necessary, a high power of the microscope must be used, and that under which the observations were made is a $\frac{1}{6}$ apochromatic with number 10 eye-piece, having a magnification of 600 diameters. This is a dry lens, and for examining fresh fluid, and especially blood, the advantages of working with a high power which exerts no pressure or traction on the cover-glass are obvious.

[In the preceding account as many plausible objections as could be thought of have been raised in order to anticipate the usual run of critical remarks. While having no doubt about the existence of the zooid, I have not been able, as yet, to ascertain if it possess a specific pathogenic action, or be merely a harmless parasite. I need hardly point out that it is a far easier task to prove the presence of any object than to satisfactorily demonstrate its absence.

I think that one of the principal difficulties in making observations on the zooid, which from the character of its movements and its general appearance seems to belong to the animal world, arises from its proneness to hide itself among the corpuscles. When the layer of blood is very thin, so that the corpuscles are far apart, the zooid is easily seen.

I have minimised as far as possible the results obtained from staining, but I am able to show a few bodies which retain their living appearance and contour.—R. G. H.]

SUBSTANCE OF A CLINICAL LECTURE ON CONCOMITANT SQUINT.

By Mr. COWELL.

It is a noticeable fact that the operation of tenotomy for strabismus has become of late years less frequent. In years gone bye rarely a month passed without the performance of several of these operations even in this a general hospital, whilst now there are comparatively few tenotomies in the course of a year. This change has been due to two facts: 1. The development and refraction of the eye are better understood; and 2. The effects of tenotomy as manifested some years after the operation have been more carefully studied.

Hypermetropia and hypermetropic astigmatism, often unequal in amount in the two eyes, and perhaps also we ought to include mixed astigmatism, are the chief causes of convergent concomitant strabismus. Donders gives the proportion due to these causes as about 75 per cent. of all the cases of convergent squint. Myopia and myopic astigmatism are the chief agents in producing divergent concomitant squint, the proportion here being 66 per cent., or two-thirds of the whole. The remainder, 25 per cent., of the cases of convergent squint will be found to be due, some to paralysis or paresis of the external rectus, or of either of the oblique muscles, and some to corneal opacities; whilst quite one-third of the cases of divergent strabismus will be found to be due to paralysis or paresis of the internal rectus, to intraocular changes, or to corneal opacities. It may also be mentioned that very rarely convergent strabismus has been found associated with emmetropia and myopia, and divergent squint with hypermetropia. The diagnosis of these various cases is not difficult, a little care and observation sufficing to enable the surgeon to recognise them, and thus to determine the appropriate treatment.

Cases of paralysis and paresis are many of them quite curable, and where corneal opacities are the cause, the squint may be remedied and is in no way to be operated upon except in the few cases where an artificial pupil may be indicated. It will be seen, therefore, that it is important in all cases to make an accurate diagnosis as to the cause of the strabismus, because the neglect of it must lead to errors of treatment, and even to occasional tenotomies, the disastrous result of which may become conspicuously apparent after the real cause of the squint has disappeared.

The way in which hypermetropic conditions of the eye produce convergent strabismus is easily explained. With the effort of accommodation is associated a proportionate effort of convergence, and it follows that in the majority of hypermetropic eyes, where much accommodation is required for distant vision, and therefore excessive accommodation for near vision, the convergence will be in excess, and clear vision in one eye will be obtained at the expense of a squint with the other. It is the production of strabismus by this associated effort in the two eyes that gives this form of squint the name of concomitant. The parallelism of the axes of the eyes is disturbed, and the relative obliquity is maintained in all positions of the eyes. It matters not which eye the patient fixes with, the amount of squint will remain the same. With this form of squint the field of fixation remains normal in both eyes.

This form of squint often shows itself very early in life, so early that surgeons often used to speak of congenital strabismus. It is a question, however, if it ever shows itself before the child begins to notice objects, and then with much error of refraction it rapidly becomes apparent. At the beginning it shows itself only occasionally, and is said to be *periodic*. Then as the eyes are more used the strabismus becomes more persistent. At first the acuteness of vision is equal in the two eyes, and the squint is said to be *alternating*, the patient being able to fix an object with either eye with equal facility, the other eye appearing to do all the squinting. After a time, and especially if there is a slight defect, such as a nebulous cornea, or a higher error of refraction in one of the eyes, the patient naturally begins to prefer the eye which possesses the more acute vision, and which can be used with the least conscious strain, and always to fix

with it. The strabismus becomes *fixed*, and the acuteness of vision of the squinting eye deteriorates.

Another important point to be mentioned is that even from the first beginning of the strabismus the patient is rarely troubled with double images, as is so often the case, for a time at all events, in paralytic squint. This is owing to the power of mental suppression, which usually is rapidly acquired in cases of concomitant squint. This suppression is of course assisted by the difference of distinctness of the images in the two eyes, the image produced in the fixing eye being on the yellow spot and therefore perfectly distinct, the image in the squinting eye being produced on the retina away from the yellow spot and therefore more or less blurred and indistinct.

In some cases of hypermetropia the asthenopia or pain and discomfort produced by the strain of accommodation and the effort to establish a balance between accommodation and convergence, continues for some time before a strabismus results. These symptoms are often at once relieved to a considerable extent on the occurrence of strabismus, exactly in the same way that they are relieved by closing one eye. In either condition the strain is at once diminished, as accommodation can be exercised without any effort to establish parallelism.

The way in which myopic conditions of the eye produce divergent concomitant squint may be similarly explained. Little or no accommodation is required for near vision in these cases, and the effort to be made is to produce convergence for near objects without it. This effort sometimes produces a considerable amount of asthenopia, symptoms which are instantly relieved by the occurrence of strabismus or by covering up one eye, in both cases the necessity for using convergence at once ceasing, and the pain and discomfort induced in making the effort disappearing also.

It must be remembered that the power of convergence is more independent of accommodation than accommodation is of convergence. For this reason asthenopia and squint less frequently result from myopia than from hypermetropia, although myopic eyes are certainly not less numerous than hypermetropic eyes, and it may be added that when divergent strabismus does occur in myopia, it does not show itself nearly as early as does convergent squint in hypermetropia.

If no treatment be adopted to restore binocular vision in these cases of squint, the eye which has begun to deteriorate spoils more and more. Generally speaking headaches, pain in the eyes, lachrymation, and various difficulties in using the eyes lead the patient, or the patient's friends, to seek advice and assistance. And if this is not obtained, or if for some reason or other the surgeon fails to afford relief, the whole retina gradually participates in the deterioration and the power of vision diminishes. In some cases the sensibility of the *macula lutea* will decrease and its predominence will be lost, the eye moving about in search of some more active point of retina, and producing in a greater or less degree the movements which constitute a form of *nystagmus*. In all the cases the eye will become more or less blind and useless. The good eye too by no means always escapes. It often deteriorates in consequence of the asthenopia produced by its use. It is therefore of the greatest importance that steps should be taken in these cases as early as possible to correct the errors of refraction on which the strabismus depends, and it is of the utmost importance if possible to establish binocular vision.

If I have made clear to you the sequence of events in the production of concomitant squint, and its subsequent effect upon the vision and usefulness of the eyes, we shall be prepared to discuss the question of tenotomy, to which I alluded at the beginning of the lecture. In all but the slightest cases of convergent concomitant squint, it used to be the custom to recommend a tenotomy of the internal rectus of one or both eyes. The rule used to be that the double operation was undertaken when the convergence exceeded two lines as measured by the strabismometer, a more convenient method of measurement to my mind than by the perimeter. My friend Dr. Edouard Meyer of Paris has invented an ingenious double strabometer for this purpose, but the single strabismometer of Lawrence is sufficient for accurate measurement if properly used. If the strabismus did not exceed two lines the tenotomy of one internal rectus, that of the squinting eye, was considered sufficient. Convex glasses were ordered to be used, some surgeons being content to correct the manifest hypermetropia or a little more, others thinking it important to ascertain and correct the whole of the hypermetropia, latent as well as manifest. Of course the astigmatism, if any, had to be estimated and corrected in the same manner. The immediate effect of this treatment was

often satisfactory. Sometimes a little divergence followed the double operation, but the patient was dismissed with the instruction to wear the glasses and persevere.

I do not think that medical men often see their own failures, but they do see the failures of others. And from these failures the inference that ought to be drawn is that we ourselves have not been free from them, and that our failures must have been seen and criticised by others. In this matter of tenotomies for strabismus many years must usually elapse before the real result is known, before the complete effect of our operation can be ascertained. Any surgeon who has been in the habit of prescribing glasses for hypermetropic eyes can hardly have failed to notice that the relief of the asthenopia, the cessation in fact of all the straining of the eyes, which results from the constant and careful use of the glasses is not infrequently followed by the gradual growth and more complete development of the eyes and a diminution of the hypermetropia. The patient grows, and the eyes freed from all their difficulties grow in slightly increased proportion. The glasses can gradually be made weaker, and in some cases, by the time that the patient is grown up, the glasses may become necessary only for reading small type or the finer kinds of work. I have had numbers of instances of this in years gone by, and now amongst my patients are the children of some of them who are going through exactly the same gradual process of glasses and development. I may even add that slight cases of hypermetropia will sometimes recover even without any treatment except attention to the general health. These patients do not suffer from asthenopia, and they may be said to outgrow the temporary deficiency.

Now precisely the same thing that I have been describing sometimes happens with our strabismus cases. After the tenotomy and the use of proper glasses the eye develops, the hypermetropia diminishes, and if complete parallelism was established by the tenotomy the development will most likely be accompanied by the appearance of divergence. If this is slight in amount, it may sometimes be obviated by diminishing the strength of the glasses, but not infrequently the divergence increases with the growth of the patient, and the development of the eye and binocular vision becomes impossible, and the patient again seeks relief.

I have no doubt that there have been patients on whom I per-

formed tenotomy some years ago who have later become the subject of divergent squint, although I have myself seen very few of them, but the few that I have seen have made me, of late years, more careful in adopting and graduating, if one may use the term, the operation. It has, however, fallen to my lot to see and treat a large number of patients the subject of divergent strabismus following tenotomy for convergence performed by others. I admit that this condition was much more common some years ago. Most of the patients had then been subjected to the old method of operating, often by general surgeons, in fact, many of my earlier cases had been operated on by Mr. Liston. But I still see several cases every year with divergence, less pronounced in their deformity than before, as the tenotomies have been sub-conjunctival, but with the same inability for binocular vision. I have therefore had to perform a great number of operations for the readjustment, or as it is now called, the advancement of these weakened muscles. It is not my object to-day to describe the operation that I adopt for this purpose, but I state it as a fact that an operation with this object is often necessary, and it points to a defect in our treatment of these cases of concomitant squint. I do not mean to say for a moment that it is not now a widely recognised fact, that many cases of convergent strabismus are successfully treated by glasses alone, or by glasses associated for a time with the use of atropine, but I think that it is not sufficiently recognised that in many of the cases development requiring time is an important part of the cure, and the friends of the patient, and often the surgeon too, are apt to get impatient and lose faith in the glasses as the sole method of treatment, and resort to a tenotomy to expedite the cure in cases where the natural process would in time prove sufficient for the purpose.

The chief element of success in, and in fact the object of the treatment of convergent concomitant squint is undoubtedly the restoration of the balance between convergence and accommodation. A tenotomy does this roughly and completely by weakening the muscles, on the contraction of which the strabismus appears to depend. Unfortunately the optical conditions of the eye are not permanent, and, as shown above, a time may come when the weakened muscles are inadequate to the duties they have to perform. The surgeon, therefore, desires to restore this balance if possible by optical means, which can be varied from time to

time with the changes of the optical conditions of the eye. In a few cases the proper glasses will establish parallelism at once, and in many others, although it cannot be done at once, perseverance with the proper optical correction will in time bring about the same result. It is a good plan to arrange the glasses so that the centre of each glass shall be placed to the inner side of the axis of vision of each eye. This arrangement in effect adds weak prisms with the bases inwards to the glasses. In my opinion tenotomy is rarely if ever necessary when the patient comes to the surgeon with the strabismus alternating, that is, with the acuteness of vision equal in the two eyes. Very often, however, the squint is already fixed, and the vision of one eye is inferior to that of the other. In many of these patients the strabismus commenced probably very early in life, and treatment by glasses obviously cannot be entered upon before the age of five years, or even later. In these cases, as soon as the patient is old enough atropine must be used, and the refraction of each eye accurately determined, and the necessary glasses used. If these do not at once establish parallelism, it will be necessary that the defective eye should be exercised daily for three or four periods of a quarter of an hour each. And this plan and the glasses must be persevered in for some time, the progress being tested every three or six months, and the glasses changed if necessary, and it will often be found that gradual improvement results. So long as no further deterioration in the vision occurs, this treatment may be continued for three or five years, and if there is improvement it may be continued indefinitely with every prospect of complete success. Some surgeons recommend the addition of weak prisms to the glasses, but I have seldom found them of much benefit, except as mentioned above. As soon, however, as we are convinced that the amblyopic eye is becoming more amblyopic a tenotomy will be necessary, as of course we shall have altogether failed to obtain binocular vision. The same thing may be said of those cases which come to us for the first time with marked amblyopia in one eye and a strabismus of over two lines. I think that in these cases a tenotomy is almost always necessary, as we can hardly ever succeed in establishing parallelism without it. But an important point that I desire to call attention to is that in these cases where tenotomy is necessary it is better not to attempt to correct the whole of the squint by

operation. It is important, in my opinion, to leave something for the glasses to achieve. The safest rules to follow I have found to be these:—

1. If the strabismus does not exceed a line and a half, defer the operation as long as possible, as it will not often be necessary.

2. If the strabismus measures a line and a half to three lines, it will probably suffice to do a tenotomy in the amblyopic eye only.

3. If the strabismus exceeds three lines, it will usually be necessary to operate on both eyes, but care should be taken, in dividing the tendons completely, not to disturb the surrounding cellular tissue, and thus avoid undue retraction of the divided muscles.

It is important to remember that if the tendon be divided close to its attachment to the sclerotic, as it ought to be, the muscle, if in a healthy condition, will retract, and form a fresh attachment to the sclerotic further back. By this means the muscle will be permanently weakened. If the tendon be not completely divided it will not be able to retract, and will soon become reattached to the same spot, and the effect of the operation will be nil. If, on the other hand, the tendon and muscle be separated too much from its surroundings by a too free use of the scissors or hook, the muscle will retract more than is desired, and be weakened too much. It is a matter of great nicety to obtain the exact effect desired, and it is an important element in the success of the operation.

In myopic concomitant squint there is usually deficiency of the internal rectus, and the operation that is often resorted to is division of the external rectus, with or without advancement of the internal rectus. My own experience is that the majority of patients, and especially if they come to us early with this condition, are better without operation. In myopia glasses for reading and near work are not usually sought, unless there is asthenopia or commencing divergence, and then the treatment should be the strongest glasses that the patient can read small type with, for constant wear. The spectacles can be so constructed that the centre of the glasses can be placed outside the axis of vision of each eye, with the advantage that the slight prisms thus obtained may relieve somewhat the weak internal recti muscles. In some cases, however, it is desirable to diminish the deformity of extreme divergence,

and, as mentioned above, the best operation for this purpose is advancement of the internal rectus of one or both eyes, with or without a tenotomy of the external rectus. The object of this operation is, after dividing the tendon of the internal rectus, to bring it forwards so that it may become reattached to the sclerotic at a point in front of its original position. In this way the internal rectus is strengthened, and the divergence relieved or diminished.

ON ALCOHOLIC STIMULANTS IN DISEASE AND THE INDICATIONS FOR THEIR USE.

By Mr. HOLTHOUSE.

That there are two sides to every question is true of nearly every subject on which a difference of opinion can arise, and on few more so than the so-called "Drink Question." This has lately been abundantly exemplified by the numerous letters in the "Times" on "Drink and Drunkenness."

In the following Paper I do not pretend to treat the subject exhaustively, but shall limit my observations mostly to such few facts as have come within my own knowledge, or are based on my own personal experience.

Alcoholic stimulants in health, are they necessary as beverages?

I cannot answer this question better than by quoting a "Declaration," which was drawn up in 1847, and signed by two thousand medical men residing in the United Kingdom and India. Among the other signatories were Dr. Addison, Dr. Neill Arnott, Dr. R. G. Babington, Dr. A. Billing, Sir B. Brodie, Sir W. Burnett, Sir J. Clark, Dr. Copland, Sir Henry Holland, Dr. P. M. Latham, Sir J. MacGregor, Dr. J. Pereira, and other distinguished medical men. It was as follows:—

"We, the undersigned, are of opinion—(1) That a very large portion of human misery, including poverty, disease, and crime, is induced by the use of alcoholic or fermented liquors as beverages; (2) that the most perfect health is compatible with total abstinence from all such intoxicating beverages, whether in the form of ardent spirits, or as wine, beer, ale, porter, cider, &c.; (3) that persons accustomed to such drinks may, with perfect safety, discontinue them entirely, either at once or gradually after a short time; (4) that total and universal abstinence from alcoholic liquors and beverages of all sorts would greatly contribute

to the health, the prosperity, the morality, and the happiness of the human race."

I pass over the 1st and 4th paragraphs as not coming within the scope of this Paper; but with the 2nd and 3rd I cordially agree, and if proof were needed, it is supplied by the improved health of the thousands of men and women in all classes of society who have left off the alcoholic beverages to which they were previously accustomed, and become total abstainers. 2ndly, By the statistics of insurance companies, benefit societies, and other temperance organisations, all of which agree in showing that the death and sickness rates are considerably lower among abstainers than among non-abstainers in the same rank of life. 3rdly, By the greater power of endurance and resistance to extremes of cold and heat by the total abstainers; as shown in the Arctic, the Ashantee, and the Nile expeditions; while in feats of sustained exertion, Weston's walk of 1,000 miles in 1,000 hours has never been accomplished by any non-abstainer. There are many other advantages that might be claimed for the abstainer; one of these is his recuperative power after severe injuries—a fact well known to surgeons, and an important factor in determining his prognosis and the kind of treatment he shall adopt. Another advantage which the abstainer has over the indulger in alcohol, is his greater susceptibility, and consequent quick response to the action of stimulants and anæsthetics, when these are required, and in quantities so small as would produce but little effect on a non-abstainer.

A few years ago I was asked to perform a small operation on the cook of a family whom I was in the habit of attending, to which she would not submit without chloroform. From the length of time she was getting under its influence, and the large quantity of chloroform that was inhaled, I felt sure that she was in the habit of indulging largely in alcoholic drinks, and before taking my departure I asked the lady of the house whether I was correct in assuming that this was the case. She appeared surprised at my question, but then told me, as a secret, which she begged me not to divulge to any member of her family, that the woman was a reformed drunkard, but was such an excellent cook, that her husband and daughters were all delighted with her. About a month after this, I learnt that this poor creature had had one of these periodical outbreaks to which such persons are

liable, and was so violent that a policeman had to be called in to get her out of the house.

Admitting as proved all the foregoing advantages claimed for abstainers, there may occur even among them conditions in which the temporary use of alcoholic liquors may become necessary. One of these is a state of exhaustion, by whatever cause produced, as, for example, after extraordinary exertion, when rest and food cannot be obtained at once, and a call is made on the already exhausted system for a fresh, or continuous, or final effort, as in certain athletic competitions. It is not unreasonable to believe that the fainting, and even death, which has occasionally happened in cycling races might have been prevented by the immediate administration of some alcoholic stimulant, than which nothing acts so speedily and effectually. I well remember, as a boy at school, the magical effect of only half a tumbler of beer and a small biscuit after a five hours' fatiguing walk without rest, food, or drink, and four miles more to be accomplished before we came to our journey's end. I don't believe I could have walked another mile without this timely aid.

Another condition in which alcohol may be taken with advantage is during the temporary exposure of the body to severe cold, without sufficient covering, or the opportunity of taking exercise. Here again I may refer to my own personal experience in confirmation. I was travelling outside a stage coach from London to Yorkshire—it was in 1824 or 1825—a day and a night journey in those days—and I shall never forget my sufferings from the cold just before daybreak, or the instantaneous and wonderful relief afforded by a glass of brandy and water obtained at the next inn where we changed horses. I felt the warm blood flowing through my arteries, as if a stream of hot water had been injected into the aorta, to the very ends of my fingers and toes, which were just before benumbed and cold as marble, and my whole body was at once in a delicious glow, and so I remained till breakfast time. Cold and wet combined are even more dangerous than simply a low temperature. I remember Lord Napier of Magdala, an abstainer himself, relating how on two occasions—once when his men were overtaken by a severe hailstorm after a fatiguing march across the mountains, and no soup or coffee could be heated for want of fuel; and another time when tossed about for two days in the South Pacific Ocean, and

all the fires extinguished—"the stimulus of spirits enabled them to hold out till a better time." In fine, there is evidence in abundance to prove that even in perfectly healthy individuals, if the system should by unavoidable causes be brought below par, and food cannot be obtained, the temporary use of alcoholic stimulants is necessary to ward off and prevent dangers that might otherwise occur. I say the *temporary use*, because their *continued use*, except in the smallest quantity, lowers the temperature of the body, and diminishes its power of resistance—a fact well known and attested to by all travellers and dwellers in the Arctic regions, who rely on plenty of solid and fatty food, and hot tea, coffee, or cocoa.

Under what circumstances may the occasional or habitual use of alcoholic beverages be expedient?

We have seen that, except in exceptional circumstances, no form of alcohol is needed for those in perfect health; on the contrary, they are in every way better without it. The same may be said of those who, though not quite up to the highest standard of health, may rightly be called healthy, for there are standards of health, as well as of strength and of stature, and what may be termed the normal standard proper to each individual, may vary considerably, even within the limit of perfect health. In all such, abstinence should be the rule.

On the other hand, there are individuals who have never come up to this healthy normal standard, and who, though not entitled to be considered ill, are nevertheless constitutionally weak, delicate, and below par; for some of these, the occasional or even habitual use of small quantities of wine or beer is certainly beneficial. Did time and space permit, I could adduce many examples in which improved health, as evidenced by increased weight, strength, and mental vigour, has resulted from a small glass of bitter ale or stout taken daily at dinner time. A medical man in Kennington who had asked me to see one of his patients, took the opportunity of introducing to me two of his daughters; they were fine handsome young women, not long out of their teens, about the same height and weight, as far as I could judge; but one was ruddy as a rose, the other paler and more delicate-looking; pointing to the first he said, "Now that girl (mentioning her name) has never tasted a drop of alcohol in

her life; but this (pointing to the other), I should never have reared her without."

I must now pass to the consideration of alcoholic stimulants in the treatment of disease and injury.

In the many theories which at different times have dominated the mind of our profession, and guided its professors in their practice, nothing is more striking than the fact that patients get well under the most various and opposite kinds of treatment, and under no treatment at all, and I fear it must be admitted that lives have been sacrificed that might have been saved, had a different treatment been adopted.

When I first entered the profession, and for some years after I had become an M.R.C.S. in 1833, the antiphlogistic method of treating all inflammatory diseases and many others, was taught and practised almost universally; and venesection, cupping, leeching, purging, starvation, combined not unfrequently with emetics, or nauseating doses of tartar emetic, was the routine practice of the day. "Severe diseases," it was said, "required severe remedies," and that many valuable lives were sacrificed to this routine practice we have abundant evidence; I need only mention the names of Howard the philanthropist, Lord Byron, and Cavour. "In all our cases," says Dr. Madden, "we did as other practitioners did—we continued to bleed, and the patients continued to die."* If, fortunately, they survived the treatment and recovered, the physician plumed himself on the success of his energetic measures; but if, as was too frequently the case, they succumbed, it was because they were too weak to bear further depletion. I well remember attending a lecture on Purulent Ophthalmia, delivered by Mr. Guthrie,† at the Royal College of Surgeons, in which he insisted on the necessity of venesection ad deliquium, tartar emetic till the patient was "as sick as a dog," and so weak that he could only "lie on his back unable to move hand or foot."

Not long after this I entered as a pupil of the Moorfields Ophthalmic Hospital, when a man who, from his pallid complexion, had probably been previously treated after the orthodox fashion of the day, presented himself before Mr. Tyrrell—at that

* Dicksons "Fallacies of the Faculty."

† Mr. Guthrie was an Army Surgeon during the Peninsular Campaign, and afterwards became Surgeon to the Westminster Hospital, and in 1816 founded the Royal Westminster Ophthalmic Hospital.

time the leading Ophthalmic Surgeon—suffering from the dreaded disease I had heard so much of at the College of Surgeons. To my surprise and horror, instead of adopting the only treatment which I had been taught could save his sight, Mr. Tyrrell, besides a tonic and a wash for the eyes, ordered him a mutton chop and a pint of porter daily. I watched the case, expecting that nothing short of total blindness could result from such treatment; but on seeing the patient a week afterwards, both the local symptoms and his general health had so much improved that it was difficult to believe he was the same person. This case made a great impression on me, and I had not to wait long before an opportunity arose which enabled me to put in practice on my own account the lesson I had learned at the Moorfields Hospital. A pale, sickly, half-starved middle-aged woman came before me at the St. Pancras Dispensary, to which institution I had been recently appointed Surgeon, suffering from keratitis of many weeks duration. She had previously been treated with lotion for the eyes and blisters behind the ears, and other remedies; and she made a speedy recovery, with greatly improved health, and almost perfectly restored vision, under treatment which was almost entirely constitutional—a daily allowance of meat and a pint of stout. In those days the public, taking their cue from the profession, adopted, as far as they could, the treatment then in vogue, and, not unfrequently, lowered their system by starvation and purgatives before they applied to the doctor for advice.

Such facts as those just cited, aided by the writings and practice of the Homœopaths and Hydropaths, and the criticism of Dr. Dickson in his amusing work on "The Fallacies of the Faculty," together with some able articles in Dr. Forbes' Journal and other medical periodicals, and lastly, by the teaching and practice of Dr. Todd and his disciples, brought about a revolution in practice. The pulling down system was superseded by the building up one; not that medical practice had been wrong before, but diseases, it was said, had changed their type! They were previously of a sthenic character, and required depletion, they had now become asthenic, and called for sustenance and stimulants.

Thus the lancet, "our sheet-anchor," as it was termed, had to give place to alcohol, which was enthroned on the pedestal from which the former had been deposed, and, like other deposed

monarchs, was subjected to the scoffs and jeers of its former worshippers—"it had slain more than the sword," and "its victims were numbered by thousands." It was not long after this change that alcohol became as firmly established as the lancet had formerly been, and swayed medical practice as absolutely as the latter had previously done. But its career was much shorter; the remedy was so pleasant, so indiscriminately prescribed, and so easy to procure, that patients were in no hurry to give it up, and it was often continued long after the necessity for its use had ceased to exist. Thus no small danger was incurred of converting our patients into drunkards, and one poor lady who I had under my care, attributed her first departure from temperate habits to the large quantity of wine she was induced to take by the advice of her doctor. It is not, therefore, to be wondered at that a remedy so fraught with danger, and liable to give rise to such sad results, was not long in following the same course as its predecessor, and we are now in some danger of discarding both the one and the other, and rushing headlong into the opposite extreme of giving no alcohol at all, however much it may be required. Thus, within and without the pale of orthodox or so-called legitimate medicine, I have seen at least half a dozen different systems of treating disease, each following the other in a natural order of sequence, and apparently as a direct consequence of the abuse of the one which immediately preceded it. Remedies in themselves beneficial, when used in moderation, fall into disrepute, and are thrown aside; yet I believe there is not one of them that has not done good; the harm that has resulted has been due to the indiscriminate way in which they have been applied, and the extravagant lengths to which they have been carried. I have seen suffering relieved and life saved by the lancet, and both sacrificed to its non-employment when urgently needed; and the same remark is equally applicable to alcohol. The real indication for giving or withholding a remedy has been too often overlooked, and attention been concentrated on some local disease, to the neglect of the general condition of the patient, of which the two cases of ophthalmia already described, afford good illustration.

Indications for the use of Alcohol in Injuries and Diseases.

There is only one indication that I know of, and that is the

condition of the patient. From the many injuries and diseases to which mankind is liable, he is frequently brought into a condition in which alcohol, in some form or other, is necessary either to save life, relieve suffering, hasten recovery, or restore health. Among the first of these may be named Shock, Collapse, Exhaustion, from whatever cause arising, whether severe injuries, as in many railway accidents, hæmorrhage, profuse or long-continued discharges, sea-sickness, diarrhœa and cholera, fevers, &c.

The occasions on which we are called upon to administer alcoholic stimulants to healthy and temperate persons are comparatively rare; but in certain injuries and diseases their use is more frequently demanded, and the only indication to guide us in their administration is the condition of the patient. Among these conditions the most serious are those which result from sudden and severe injury, as in railway accidents, in which there is always, if consciousness be retained, a certain amount of shock or collapse produced, a condition in which the vital powers are so depressed as to threaten the speedy extinction of life, unless some powerful stimulus be administered. In these circumstances brandy is, I believe, one of the best restoratives, our object being to tide over a crisis and keep the patient alive till the system has recovered from the shock. Whether it should be continued after reaction has set in, must depend on the condition of the patient, and be left to the judgment of the surgeon; as a general rule it should be discontinued, but this is not always expedient, and will depend very much on the previous habits of the patient. In those severe injuries in which recovery seems doubtful, but not impossible, we may sometimes be helped to a prognosis by the patient's behaviour in regard to stimulants. If he should decline to take them, or the stomach should reject them when taken, or if retained no reaction should ensue, the patient will certainly die. If, on the contrary, they are swallowed readily and retained, there is hope. Again, when from the nature of the injury recovery is manifestly impossible, the patient should not be worried with importunities to take either this or that kind of stimulant, but may be indulged with whatever he asks for, which, in the few cases of this nature that have come under my observation, is more frequently a glass of cold water.

In many diseases of a grave nature which require stimulants, such as certain medical and surgical fevers, brandy is also the best form in which alcohol can be administered, but it should always be done tentatively, and its effects watched. If it reduces the frequency of the pulse, and increases its power, its action is beneficial, and it should be continued as long as it is called for by the condition of the patient; but should the effect be so slight as to be scarcely appreciable, a larger dose is needed and should be increased until a decided effect is produced. The quantity which is sometimes required to rescue persons addicted to spirit drinking is really astounding; thus the late Dr. Carpenter, writing of a severe epidemic of typhoid fever which he witnessed in Edinburgh in the years 1836-37, mentions the case of a woman of intemperate habits, who took a bottle of sherry and 12 ounces of whisky daily for more than a week before recovery was effected.

I could mention other cases in which lives have been saved by large quantities of brandy, and where they would certainly have been lost had a less quantity been given. (See Cases 4 and 5.)

The question naturally arises, whether any precise rule can be laid down as to the quantity of brandy which should be administered in any given case, and I am bound to say I know of none. Every case must be judged by itself, and the amount to be given determined by the actual state of the patient, and a knowledge of his previous habits. Another question to be determined is, When should it be discontinued? And the answer is, Whenever the health is so far improved as to render it no longer necessary. It is a curious fact, that so long as the alcohol is really required, its action seems to be concentrated in battling with the disease and sustaining the patient; but as soon as it has done its work in this direction its intoxicating effects, hitherto in abeyance, begin to manifest themselves, and patients have remarked to me that they *then* knew they were getting better; for before, the brandy had no more effect on them than water.

There is another condition which should be mentioned in connection with this subject, *i.e.*, the thorough prostration and exhaustion, sometimes ending in death, caused by sea-sickness,

in which brandy fails, even when given with effervescing waters, but where champagne succeeds.

A striking illustration of this was related to me by a friend who suffered in an excessive degree from sea-sickness in a voyage from Rio to England. All sorts of stimulants were tried to arrest it, but without avail, and she was reduced to the last extreme of exhaustion, and all hope of her recovery had been given up, when, as a last resource, champagne was tried, with the immediate result of stopping the sickness, and from that time she gradually improved, and reached the end of her journey in comparative health.

Cases Illustrating the *Beneficial* Effects of Alcoholic Stimulants.

Case 1. *Infantile Diarrhœa.*—The patient in this case was one of my own children, a little boy between one and two years of age, who was attacked with the above disease during a very fatal epidemic of diarrhœa which broke out in London in 1857. His symptoms were frequent dejections from the bowels, great thirst, and vomiting, so that nothing given him in the shape of food or medicine could be retained. From the wasted and shrunken figure and prostration of the child, it was evident that unless something were done to stop the evacuations he could not survive many hours. Under these circumstances I called in my friend and colleague, the late Dr. Fincham, and by his judicious advice one teaspoonful only, of milk with two or three drops of brandy was given every half-hour, and under this simple treatment the purging and vomiting ceased and the child quickly recovered.

The indications in this case were plain enough, but the success of the treatment was probably due as much to the mode of administering the remedy as to the remedy itself. A larger dose would probably have been rejected and the child lost. By giving just as much as the stomach could retain, and no more, he was saved.

A case, somewhat similar to the one just narrated, is published in the "Temperance Journal" of December 2nd, 1880, by Dr. Alfred J. H. Crespi, a pronounced teetotaler, who has been honest and courageous enough to give some other cases in which

the non-alcoholic treatment failed, and brandy was obliged to be given to save life. An abstract of one of these I will give in nearly his own words.

CASE 2. *Diarrhœa.*—After premising that he had attended many severe cases of diarrhœa and dysentery without brandy, Dr. Crespi adds: "But a month since, I had a case that taught me once more caution and not to expect invariable success from any treatment, nor to think much of isolated cases. The child was apparently dying. I regulated the diet carefully, wrote from first to last four prescriptions, and was helped by a skilful and experienced surgeon of fifty years standing. A third medical man at last saw the case. He stopped all the medicines, told the parents to give the little sufferer any food or drink he liked, and, as medicine, one drop of brandy every two hours. The profuse diarrhœa speedily ceased, the child rallied, and, when I last heard from my friend, was almost well." In this, as in another case of Dr. Crespi to be presently quoted, it is evident he is sceptical as to the change for the better being due to the brandy, for he adds: "The drop of brandy could not, in my humble judgment, have had much to do with the result."

CASE 3. *Prostration and Nervous Excitement.*—Dr. Crespi's. It was a farmer's wife whom he attended in her confinement. "The woman was fearfully nervous. From the first she asked for chloroform and clamoured for brandy; her excitement at times was tremendous. After the affair was over, her condition did not particularly please me. She was faint, cold, and half insensible; sal-volatile and other remedies seemed powerless. The friends seemed anxious for a second medical man, and an able and pleasant enough man was summoned. In a few moments he proposed brandy and water, a tumblerful every hour. Brandy was given, to the relief of the friends and relatives. The woman at once complained of less pain and uneasiness and quickly went to sleep. From some cause or other, next day the patient was doing wonderfully well and soon pulled round."

Surely this was a case in which the intense excitement and nervousness and the subsequent prostration strongly indicated the propriety, if not the absolute necessity, of giving alcohol,

especially after the failure of sal-volatile and other remedies, and the favourable change in the woman's condition so quickly following the administration of the proper remedy is a sufficient condemnation of the principle which withheld it.

CASES 4 AND 5. *Two cases of Carbuncle.*—I had under my care in the St. Matthew Ward, within a few weeks of each other, two very instructive cases of carbuncle. The men were nearly of the same age—between forty and fifty—the situation of the carbuncle was the lower part of the neck, and one of them was the largest I had ever seen, extending from shoulder to shoulder.

The treatment, local and general, was very much the same in both; with this difference, that the patient with the large carbuncle was a man of temperate habits, and no indications arose for adding to the ordinary full diet on which he was placed, alcoholic stimulants being withheld, partly because they were not called for by the condition of the patient, and partly because he did not wish for them.

The other patient, on the contrary, had been fed up, and had brandy given him almost from the beginning; in spite of this he made no progress towards convalescence, but got weaker and weaker, and it was evident to all that unless some speedy change for the better should take place his life could not be prolonged many days. It was while he was in this critical condition that I visited him one morning at 9 a.m. He lay on his back exhausted and apparently moribund; his eyelids only partly closed, and the conjunctiva and corneæ dry and covered with a film; his mouth partly open, revealing gums and teeth covered with sordes; his pulse, extremely feeble and rapid, could not be counted, and he could hardly be roused to grunt out an answer to questions; it seemed very doubtful whether he would survive for more than a couple of hours.

Being in the operating theatre that same afternoon (it being the hospital operation day), I said casually to the House Surgeon, who had accompanied me to the bedside of this patient in the morning, "Well, what time did the poor fellow die?" "Die, sir," he replied, "he's better!" I stared at him in astonishment, but it was true. He then gave me the following account: "After you left this morning I poured spoonful after spoonful of brandy down his throat, and at length he rallied."

I think it will be admitted by all unbiassed men that this patient's life was saved by the brandy, administered not according to the directions of teetotal doctors, *i.e.*, in small definite quantities as if it were hydrocyanic acid or bichloride of mercury, but in quantities sufficiently large to produce the desired effect. Now I maintain that when a patient is hovering between life and death, a stimulant like alcohol ought not to be given by measure, but the quantity must be regulated by its effects. These will vary with the state of the patient and his previous habits; one ounce of alcohol given to an habitual abstainer will produce as great an effect as four times that quantity would do on one who indulges freely in daily potations of alcoholic beverages, just as habitual opium eaters will consume large quantities of that drug to produce the same effect as an ordinary dose on a person who is unaccustomed to the drug.

Besides the benefit derived from the administration of alcohol in the grave conditions we have already described, there are some others in which I have also witnessed good results. I will merely enumerate three of these.

CASE 6. *Ununited Fracture, or rather delayed union, of the Tibia and Fibula.*—A young man was under my care in 1832, when dresser to the late Sir William, then Mr. Lawrence. The patient had been in bed six weeks with his leg in splints; the bones were in perfect apposition, but no consolidation or union had taken place. The man having been on low diet all the while, as was the custom in those days, and looking pale, though feeling well, Mr. Lawrence put him on full diet, with half a pint of beer daily at dinner. This change of diet was soon followed by a change in the condition of his limb, and three weeks after he was allowed to leave his bed and go about on crutches.

CASE 7. *Tic Doloureux.*—This was related to me by the sufferer himself—a Nonconformist minister—formerly a strict abstainer. He said he had suffered from the affection for many years and had tried various remedies but without permanent benefit, till some friend advised him to break his pledge of total abstinence, for a time at least, and try porter. This he did, and with such good effect that he was completely cured of the neuralgia, ceased to renew his pledge of total abstinence, and became what a

facetious clerical friend called an example of a "Reformed Teetotaler."

CASE 8. *Obstinate Gleet.*—I have met with two cases of gleet of some months duration, and a third is related by Bransby Cooper in a clinical lecture delivered to the students of Guy's Hospital, in which this troublesome disease was suddenly cured by a drinking bout. In each case, the youths, who had previously been in the habit of using alcoholic beverages, left off taking them when they contracted the gonorrhœa, from a belief that they would retard their recovery.

I have treated hitherto of certain conditions in health, disease, and injury, in which the administration of alcoholic stimulants was either imperatively required to save life, or to restore and improve health. I have now to give a few examples in which they are hurtful.

CASES ILLUSTRATING THE *Injurious* EFFECTS OF ALCOHOLIC STIMULANTS.

CASE 9. *Arthritis of the Left Elbow Joint.*—This case involved the skin and all the soft parts intervening. I cannot give more particulars of the case or of the treatment, as I have no notes I can refer to, but the fact which I wish to impress on the reader is the striking improvement which followed a change of treatment. I was annoyed at the obstinacy of the disease to the treatment adopted. The local symptoms were at a standstill, and noticing the man's flushed face, ferrety eyes, and irritability of temper, I asked for the diet card, which I had not looked at after his diet had been ordered on admission, and then discovered that a large quantity of brandy had been ordered by the House Surgeon, in addition to the beer which had been allowed when the diet was set. In spite of many protestations from the patient that he could not live without the brandy, that it was the only thing which kept him alive, &c., &c., it was forthwith discontinued, and the improvement both in the local disease and his general appearance and manner was very remarkable and highly satisfactory.

CASE 10. *Chronic Ulcer of the Leg.*—A pleasing looking well-

mannered woman, who had been a cook in some good families, and had a rather *bon vivant* face, came under my care in Chadwick Ward for a large chronic ulcer on the lower third of the right leg. The only feature in the case deserving of remark was the alternate improvement and deterioration of the condition of the ulcer, the cause of which I did not for some time discover, but it was at length explained by some of her friends being detected in bringing in brandy.

The most remarkable instance that I know of as illustrating the advantage of abstinence in ulcers of the leg is published in the "Bristol Temperance Herald," of which I here give a short abstract.

CASE 11. *Ulcer of the Leg.*—The lady resided in Bristol, and until her 89th year, had daily taken for the larger portion of her life a beverage of some kind of intoxicating drink, viz., beer, the best Madeira wine, and occasionally gin and water. This practice she relinquished all at once in the 89th year of her age. For a time it was feared her health would suffer, but far from this being the case, it improved. For nearly 15 years she had had an ulcer on one of her legs, which at times was troublesome and appeared dangerous. After practising abstinence for about a year and a half, it began to diminish, and was soon perfectly healed. At the expiration of two years she had a rather violent attack of influenza, which brought her so low that her medical attendant recommended wine, which she took for about six months, when the wound in her leg again opened and became troublesome. The wine was consequently discontinued, and after the lapse of a few months the sore again healed. Her health also improved more decidedly than after her first trial of total abstinence, and she continued until a few days of her death, at the age of 93, in the enjoyment of excellent health and spirits.

REPORTS OF SPECIAL CASES.

CASES ILLUSTRATING THE INFECTIOUSNESS OF LOBAR PNEUMONIA.

By Dr. WILLS.

The three following cases occurred in King William Ward in February, 1891, under Dr. Sturges. They are reported as illustrating the infective nature of certain forms of pneumonia. Of this we had several illustrations in the hospital during the epidemic of pneumonia which occurred during the first six months of 1891, coincidently with the widespread epidemic of influenza occurring during April, May, and June.

George M—, æt. 45, a labourer, was admitted on February 2nd. He had been a sailor in the Navy for eleven years, and had also served some years in the American Navy and the merchant service. He had been a heavy drinker when abroad, but not lately. Eight years ago he had pleurisy and inflammation of the lungs. Winter cough for the last eight or nine years.

On January 31st he shivered and had pain in the left side of his chest. On February 2nd, when admitted, he was slightly delirious and very sleepless. Cough troublesome and frequent, expectoration scanty and rusty. Sweating profusely. Tongue dry and rather brown. Temperature 100·6°, rising next day to 103·2°. Pulse 132. Heart sounds normal. Respirations 30. Resonance good all over both backs, except at extreme left base, with numerous moist sounds, but no tubular breathing. Dulness at left apex anteriorly, with distant bronchial breathing and a few crepitations. Urine, no albumen. From the 3rd to 6th the temperature gradually fell to normal. The bronchial breathing at the left apex became more marked, and more numerous and larger moist sounds were heard. Expectoration became more profuse, but ceased to be rusty. His general con-

dition remained that of great prostration, with slight delirium. The delirium continued for five days after his temperature became normal. The pneumonic signs at the left apex had disappeared by the 12th, and he rapidly improved, and was able to get up on the 16th.

On the 19th, however, his temperature again rose to 102·4°, and he became again delirious. Dulness, bronchial breathing, and crepitations again appeared at the left apex. His respirations rose from 24 to 36, and then to 40. He rapidly became weaker. On 22nd severe pain came on in right iliac region, and lasted till next day, when he died. There was no albumen in the urine throughout his illness.

In the next bed to this man, on the right, was a patient named James S—, a gas-stoker, æt. 38, who was admitted on January 31st with severe diarrhœa, and hæmorrhage from the bowel. He had been a very heavy drinker, but had had no previous illness except influenza in 1890. The diarrhœa had commenced three weeks before admission, coming on while he was lifting a heavy weight. His temperature was normal, and his chest was perfectly healthy when admitted. The diarrhœa, being apparently due to some ulceration of the colon (found post-mortem), does not need further mention in relationship to the attack of pneumonia. On February 5th, till which day he had been kept in bed and was perfectly free from chest symptoms, he vomited, and his temperature rose to 104°.

For the next three days he was frequently sick, but no physical signs of pneumonia could be found, although his respirations had risen on the 7th to 48, and his chest was carefully examined. On the 9th dulness and bronchial breathing were found at the right apex. The patient became delirious, and the temperature rose to 104·8°, having remained between 103° and 104° since the 5th.

On the 10th his temperature fell to normal, but his condition did not improve; the physical signs of pneumonia became more marked; he rapidly got weaker, and died at 1 p.m. on the 11th. On the 9th albumen was found in his urine. He did not expectorate at all.

In the next bed but one to George M—, but on the left, was a patient named Frederick B—, a barman, æt. 29, who came in on January 15th with chronic Bright's disease

(parenchymatous nephritis). He had also been a heavy drinker. Except rheumatic fever eight years before he had no other illness. His renal symptoms dated from Christmas, 1890.

His condition on admission was the usual one of a patient suffering from chronic renal disease. There was some œdema of the scrotum, but none of the legs. Urine contained $\frac{1}{8}$ albumen.

There was some impairment of resonance at the left base, and bronchitic sounds were audible all over the chest. Expectoration was profuse and blood-stained. Temperature varied between 100° and normal. By February 6th he had much improved; his temperature was normal, and he had almost entirely lost his bronchitis. On February 9th he shivered, and his temperature rose suddenly to 104·6° and he was sick. His temperature fell that evening to 100°, rose again next morning to 104·8°, fell on the 11th again to 99·2° and he sweated profusely, but it rose again on the 12th to 105·2°, and from the 13th to the 15th remained between 104° and 102°, falling gradually on 16th and 17th to normal. The physical signs of pneumonia were not evident until the 15th, when there was found dullness, bronchial breathing, and numerous crepitations at the right apex, but throughout the man had the appearance of a patient suffering from pneumonia, and his pulse respiration ratio averaged 3—1. There was no herpes at any time. During the attack his albuminuria increased and he was extremely ill, and very nearly dying, but from the 17th he gradually improved, and went out convalescent. The delirium continued for two days after his temperature reached normal.

It is difficult not to believe that these two latter cases had in some way their origin in the attack of pneumonia of George M—. Certainly to those who saw them at the time there seemed a strong probability of a causal relationship.

George M—'s pneumonia was an unusual one. He had a relapse, he was delirious after his temperature became normal, and the part of the lung affected was the apex.

In both the latter cases the apex was also the seat of the inflammation. The case of J. S— was the most striking because he was not suffering from a disease in any way predisposing to pneumonia, whereas Fred. B— was the subject of Bright's disease.

M— came in on February 2nd. S— was attacked on

the 5th, when M—'s pneumonia was at its height. B— was attacked on the 9th, when M—'s temperature had fallen to 99°, but he was still delirious and still expectorating rusty sputum, and therefore still in a perfectly fit state to act as a source of infection.

I am not aware that any communication took place between M— and B—, but as M— and S— were in neighbouring beds, it is extremely probable that they occasionally used the same spitoon, if S— used a spitoon at all; and M— being delirious, may very likely have drunk out of S—'s mug of milk, but of that I have no evidence. There was nothing unusual noticed in the post-mortem appearances of the lungs of either M— or S—.

As corollaries to these cases, I may mention the following instances, which occurred during the recent outbreak of pneumonia, of persons who seem likely to have infected each other or derived their pneumonia from a common source of infection.

Louisa D—, æt. 13, was admitted on February 14th under Dr. Donkin with pneumonia at left apex. Her father was lying dead in their house at the time of her attack.

Kate C—, æt. 43, was admitted on March 12th with pneumonia at the apex of the right lower lobe. Her pneumonia ran a very protracted course, and the temperature subsided by lysis. She was unusually prostrate in her convalescence. On March 21st her husband, æt. 44, was admitted under Dr. Sturges with left basic pneumonia. His pneumonia had commenced six days before admission, was of the ordinary type, and ended by crisis. He had been nursing his wife all night and working by day, and had become, in consequence, much exhausted.

Out of eighty-one cases of lobar pneumonia admitted this year up to October, besides the six cases referred to above, in six other cases there were bad smells in the house, or the drains were in process of repair. In 43 cases no cause in the way of chill or poisoning could be ascertained, leaving thirty-eight cases in which chill (very trivial often) or getting wet through was suggested as the cause. If against those thirty-eight cases we put the twelve in which infection from another case or sewer poisoning was possibly the cause, we obtain a very high ratio for the infectious cases in the recent epidemic.

I use the term epidemic advisedly, as the [illegible]alence of

pneumonia during the first six months of 1891 fairly deserves to be considered an epidemic; witness our admissions during this period—76 in number, against an average for the same period of the last six years of 30·3. And in this connection one must not forget that it was during this same period that the second influenza epidemic was rife. However, with regard to the influence of an influenza epidemic on the admissions for pneumonia it is only fair to say that in the first six months of 1890, when there was also an influenza epidemic, the number of cases admitted was only 28.

In Drs. Sturges' and Coupland's "Pneumonia" several instances from both British and foreign sources are quoted of outbreaks of pneumonia spreading by contagion in a household or a village, some of which are extremely striking. But it is in answer to the following sentence in the part devoted to the question of etiology that I have recorded these three cases, though I fear that they only answer the requirements of the first half of the sentence and not the second:—

"What is required for proof of real conveyance is this, that the affection should spread when into a place hitherto free of it; and that this should occur with sufficient frequency to override accidental coincidence."

TWO CASES OF PULMONARY EMBOLISM, WITH REMARKS.

By Dr. HEBB.

Case 1.—E. W., female, age —, was admitted under Mr. Davy on February 7th, 1889, for internal hæmorrhoids, from which she had suffered for the past three years.

Mr. Davy operated on February 12th, the piles being removed by the "clamp and cautery" operation.

The patient seemed to be going on fairly well till February 28th, when soon after midnight she complained of feeling ill, dyspnœa being the most urgent symptom. After a while the feeling of oppression and the sensation of dyspnœa somewhat abated, but at 7 a.m. she "shifted in bed, turned over on her side, and died in a few minutes."

The post-mortem examination was made thirty hours after death. Body well nourished. Varicose veins and scars of ulcers on right lower extremity. The varicose veins feel like knotted cords, and much of the skin is pigmented. The right leg and thigh are swollen, but there is no subcutaneous œdema.

The whole of the veins of the right lower extremity are distended with firm black clot, adherent to wall of femoral vein, and reaching upwards for about 1 inch above Poupart's ligament.

Bladder much distended, reaching about 2 inches above the umbilicus.

Uterus and ovaries normal. Gastro-intestinal mucosa seems normal throughout, there being nothing specially noteworthy about the rectum. Liver 72 oz., spleen 8 oz., kidneys 8 oz.

Pleuræ normal. Lungs very œdematous. The larger branches of the right and left pulmonary arteries are filled with firm clot. From the right was withdrawn a plug about one inch long. It is rounded at its inferior (cardiac) extremity, and adherent to a

less coherent and black clot. This plug consists of firm and partially decolorised fibrin. It is obviously an embolus, and is probably derived from the clot in the large vessel of the right lower extremity. The right auricle, ventricle, and pulmonary artery contain some decolorised clot, similar to that which is constantly found in this situation, and which differs altogether from that which is described as being an embolus.

Heart 11½ oz. Endocardium normal. Valves competent. Wall of right heart very thin near apex, much subepicardial adipose tissue.

CASE 2.—C. H., male, age 36, was admitted under Dr. Donkin on August 20th, 1891. Is a shoemaker living at Brixton. Family history unimportant. Personal history: twelve years ago was laid up for four months with acute bronchitis; since then has had occasional attacks of quinsy, but no other illness until December, 1890, when he was attacked with influenza, and of which he had a relapse in January, 1891. This last illness was accompanied by bronchitis and dyspnœa, and he had to lay up for a week. The cough and dyspnœa have continued ever since. About six weeks before admission his right foot and leg began to swell, and soon afterwards the left leg. Three weeks ago he expectorated some blood, bright red at first, afterwards becoming darker. Six days before admission the lower half of the left side of his face was suddenly paralysed, and his speech became difficult.

On admission was slightly jaundiced; was obliged to sit up, as on lying down his breath became very short.

Slight paresis of left lower face. Tongue protrudes quite straight. Speech only slightly thick. Complains of pain all over right chest and abdomen.

Chest movements impaired on right side, dulness over lower half of right thorax. Rhonchi all over chest, and some crepitations audible in left mammary region. No cardiac dulness. Apex, obscurely felt in mid axillary line at sixth interspace, heaving in character. Heart sounds feeble, muffled.

Pulse 116, small, hard, wiry. Respirations 40. Œdema of feet and legs. Urine contains $\frac{1}{10}$—$\frac{1}{12}$ albumen.

The patient continued in much the same condition, suffering greatly from dyspnœa. The œdema gradually diminished. On

September 6th was suddenly seized with urgent dyspnœa, and died in a few minutes.

Post-mortem examination made fourteen hours after death.

Body emaciated, moderate but universal jaundice. Anasarka. Ascites.

Brain 45 oz.; membranes and nervous tissue normal. Spinal cord normal.

Fauces and larynx nil.

Right pleural surfaces over the whole of the lower lobe are adherent by the intermediation of a layer of soft yellow fibrinous lymph, the subjacent tissue being hyperæmic. The rest of the pleura is unaffected, the sac containing quite a pint of clear watery fluid. Over the left lung are a few old adhesions, and much fluid in the sac. Both lungs are very œdematous. In both lower lobes are hæmorrhagic extravasations, which occupy almost the whole of these lobes. At the lower part of the left upper lobe are two infarct areas, yellow in colour with irregular wavy borders, each about the size of small walnuts. One of these has softened in the centre and broken down, the other is quite firm. The hæmorrhagic infarction is much more intense in the right lung than in the left. From both right and left pulmonary arteries were extracted granular decolorised fibrinous clots by which those vessels were plugged, and which in every respect resembled the clot in the femoral veins and some of the clot found in the right ventricle.

Excess of fluid in pericardial sac. Heart 17 oz. Both ventricles hypertrophied. Endocardium normal. Pulmonary and aortic valves competent. Both sides of the heart contain much soft black clot. At the apex of the right ventricle, partly free and partly entangled among the columnæ, are some pieces of firm decolorised granular fibrinous clot.

The main veins of both lower extremities are filled with firm decolorised clot, which is adherent to the walls of the vessels, and extends upwards into the large veins of pelvis.

The interna of aorta is swollen in places, and from these it can be stripped off cavity.

Liver 65 oz., spleen 13 oz., kidneys 6¾ and 7¼ oz.

These viscera and the gastro-intestinal mucosa are in that condition which frequently results from stasis of blood and imperfect circulation, but in other respects are not noteworthy.

Remarks.—These two cases have one significant factor in common, namely, that of the thrombosis of the femoral veins. In both cases the clot reached into the pelvis. The end of the clot breaks off, and is transported first into the heart and afterwards into the main branches of the pulmonary artery. Death from shock and asphyxia. Thus the mechanism of the transaction is simple enough.

In the first case the formation of the clot evidently was closely associated with the previously existing varicose veins, and the clotting may be assumed to have commenced, or at any rate extended, after the operation for removal of the hæmorrhoids.

The thrombosis is unilateral, and therefore, especially when taken in connection with the varicosity of the subcutaneous veins, was of local origin. But in the second case the condition was a general one; the veins of both extremities were thrombosed; the thrombosis began practically simultaneously on both sides. The condition of the viscera, *e.g.*, the liver, the spleen, the kidneys, showed that they had been subjected to a slow circulation and condition of partial stasis for some period.

This is further accentuated by the hypertrophy of the heart, and the hæmorrhagic extravasation and congestion in the lower lobes of the lungs.

It is to contrast the conditions of the lungs, and to point out that the hæmorrhagic infarct areas are not connected with emboli, but with the inability of the right side of the heart to force the blood through the lungs, and of the left side to receive a further supply. The consequence of this state of things is that the lungs get gorged with more blood than their vessels will contain, and then they burst in their weakest place. The result is hæmorrhagic extravasation and infarct. This state of things, brought about by precisely similar pulmonary and cardiac conditions, is of quite frequent occurrence, and must be familiar to every one, but the explanation is usually, I think, conferred on embolism.

The condition produced by embolism is quite different, and the characteristics of the results of the two events are well shown in Case 2. In the left upper lobe two emboli, from the venous clot, evidently had been lodged, and at different times. The wavy, irregular yellowish border, surrounded usually by a hyperæmic zone, encloses an area which is at first pallid, and afterwards most

usually becomes quite yellow; which is usually located at the periphery of an organ, and the surface of which becomes eventually depressed at the embolised site; wherein in microscopical sections the skeletons of the tissue of the embolised area can always, at first at least, be descried; wherein there is no evidence of any hyperæmia or extravasation; and which may become softened in the centre, but probably most often does not. These are the contrast marks between the results of an embolus and those of congestion-extravasation, and it is useful to bear in mind that the hæmorrhagic infarct, the congestion-extravasation, occurs as the accompaniment of conditions which almost entirely preclude the possibility of embolism.

In the first case there was no trace of engorgement of the lungs. The lungs were very œdematous. The moribund condition lasted seven hours, and it is quite possible that one lung was embolised before the other.

A comparison of the contents of the cardiac cavities is interesting. In the first case the moribund condition lasted about seven hours, and in the right side is found a decolorised clot which reaches into the pulmonary artery. This kind of clot, the clot of the moribund, is of constant occurrence, and varies very much with the disease and with the duration of dying (moribund state). In this instance the moribund state lasted about seven hours, the amount of white clot in the right cardiac cavities therefore took about seven hours to form.

In Case 2 the clinical record states that the patient died quite suddenly. The contents of the cavities of the heart tell the same story. They were filled with soft black clot; the "moribund" clot was not there. And these two cases therefore illustrate the quality of blood clot found in the heart when death has been slow and when it has been sudden.

In connection with the foregoing cases I may further illustrate the position from a lung which I have inspected since writing these notes. As the case did not belong to Westminster Hospital I can only just allude to it.

In this instance the pulmonary artery and its larger branches are choked with firm decolorised adherent clot. But there is no extravasation, no hyperæmia, no engorgement. Quite the contrary. The lung appears smaller than normal. This is just as it should be, whenever of course there is no obstruction ahead.

For the block in the main pulmonary artery prevents any further access of blood, and does not prevent the blood in front of the block from draining away.

In conclusion, I may point out that an interesting feature in Case 2 is the fact that the patient had influenza, with a relapse, or possibly that exacerbation to which this disease is so liable, and that he never really recovered from the effects of this disorder.

A CASE OF DELIRIUM TREMENS, ILLUSTRATING ONE OF THE CAUSES OF CARDIAC FAILURE IN THIS DISEASE.

By Dr. HEBB.

D. B., male, aged 30, was admitted on September 10th, 1890, under Dr. Sturges for an epileptiform attack, apparently the result of alcohol.

Had been a soldier, but recently had become a gas stoker. For the past four or five years had suffered from winter cough. There was no history of syphilis. Was a heavy drinker, and had had four or five previous attacks of delirium tremens, and also two fits similar to but less severe than that for which he was admitted to Westminster Hospital.

There is a strong family history of alcoholism. One sister died from effects of drink.

He was taken ill on the night of September 9th, and when admitted was suffering from great dyspnœa, the surface of the body being cold and the face livid. Respirations 44. Examination of the lungs only revealed a few scattered moist sounds. Pulse 108, regular, but almost imperceptible. Pupils equal, but very sluggish. The urine contained about ¼ albumen and granular casts. The next day, September 11th, was still cyanosed, and the temperature subnormal. Was restless, suspicious, and in the condition of general tremor indicative of delirium tremens.

Towards evening became gradually more violent, and died suddenly at 12.30 a.m., September 12th, 1890.

Post-mortem examination (see P. M. Book, vol. vi. p. 128) was made fourteen hours after death. Body well nourished. Tissues and fluids in serous sacs much blood-stained.

Brain 43 oz. Skullcap very thick. Excess of subarachnoid

fluid. Pia opaque in places, and adherent to cortex about sylvian fissures and at the base. It is not thickened. The nervous tissue is flabby. It is not injected.

Larynx and fauces nil. Right pleural surfaces universally adherent. Considerable excess of fluid in left pleural sac, the lower half of the lung being compressed thereby. Both lungs are very œdematous, but in other respects not noteworthy.

Pericardium normal. Heart 18 oz. Muscle soft and pallid, but in many places is mottled with intramuscular petechiæ. The petechiæ are most numerous in the columnæ carneæ. Valves competent. Endocardium smooth and shiny. Aorta normal.

Liver 90 oz., soft and pale but not yellow, and with a faint nutmeggy appearance. Spleen 6½ oz., flabby, pale. Kidneys 8½ and 9 oz., firm, reddish, smooth. Cortex relatively large. Cardiac end of stomach thinned from cadaveric softening. Omentum adherent to symphysis pubis. Diverticulum of ileum about 2½ inches long.

Microscopical examination of heart muscle.

Transverse sections of columnæ carneæ show largish areas where the fibres are separated by granular fibrinous débris, and others where there is extravasation of red corpuscles. In some situations the vessels are crowded with leucocytes, while in others the red disks preponderate.

The coats of the vessels, especially of the branches of the coronary artery, are thickened.

Remarks.—The microscope was able to show the cause of the cardiac failure. The heart gave way from general feebleness, and this weakness seems to have affected it throughout, although the columnæ carneæ were the parts most severely damaged.

Although the sections showed that there was extravasation of the contents of the blood-vessels, the impression imparted to me, after carefully inspecting the general appearance as well as the details of the microscopical preparations, was that the lesion was not primarily vascular. It seemed far more likely that the weak state of the cardiac tissue generally was to be regarded as the principal factor, and that the rupture of the vessels was an accompaniment or secondary phenomenon.

TWO CASES OF "PERFORATING" ULCER OF THE DUODENUM, WITH REMARKS.

By Dr. HEBB.

Case 1.—S. P., female, aged three months; was admitted under Dr. Sturges on October 29th, 1891.

Was born at full time, has been fed from the breast only, and has had no previous illness.

Family history unimportant, but it may be mentioned that her brother, aged two years, was in the hospital at the same time for broncho-pneumonia.

The patient was in good health until October 24th, when she began to ail, the chief symptom being apparently diarrhœa. On October 27th the mother noticed a red rash on buttocks; the rash was merely a redness, there being no swelling or tenderness. The next day the red surface became sore, and bled when washed.

When admitted the infant seemed very ill. Temperature 100°. Vomited several times daily. There was slight retraction of the head, and general rigidity, with clenching of the hands and flexing of the thighs. No abnormal physical signs in thorax or abdomen. There are intestinal evacuations seven or nine times daily, the motions being light yellow and watery. They were not offensive.

The vomiting continued at frequent intervals. On November 5th passed a little blood per anum, became blue, and died soon after.

Post-mortem examination (see P. M. Book, vol. vi. p. 400) was made seventeen hours after death. Body well nourished. Sores on buttocks and on right flank just above middle of iliac crest. The margins are irregular, bluish in colour, edges shelving. The ulcerations are comparatively superficial, reaching only about as far as the deeper layers of the epidermis. The area of skin involved would be about 3 square inches.

On opening the abdomen several teaspoonfuls of viscid slimy brownish fluid were found between spleen, stomach, and transverse colon. There is no trace of peritonitis. On removing the gastro-intestinal tract two ulcers are found on the posterior aspect of the duodenum: one of these, measuring $\frac{4}{16}$ by $\frac{3}{16}$ inch, lies quite close to pylorus; the other, $\frac{3}{16}$ by $\frac{2}{16}$, is about half an inch from the ring. The former is a clean punched-out hole, and involves all the coats of the intestine. Its internal circumference is a little larger than its external. The second ulcer is a shallow pit reaching to about the middle of the deeper layer of the mucosa. On the anterior surface of the duodenum, close to the pylorus, is a circular patch of capillary hæmorrhage into the mucosa. The diameter of this patch is about $\frac{3}{16}$ inch. There are a few small hæmorrhages into the gastric mucosa. With these exceptions, the gastro-intestinal mucosa is quite normal. The stomach contains about half a drachm of thin reddish-brown fluid. There is no foreign body in the gastro-intestinal canal. The fæcal contents are thin, yellow, and almost fluid. There are no enlarged lymphatic glands in thorax or abdomen. The reaction of the contents of the small intestine is intensely acid. The thoracic and abdominal viscera (except duodenum and stomach) are perfectly normal, and appear quite healthy. Brain ($18\frac{1}{2}$ oz.) and spinal cord normal.

CASE 2.—J. H., male, aged 63 years, a blacksmith, was admitted on August 19th, 1891, under Dr. Sturges. No previous history was obtainable, except that for the past four weeks he had been subject to attacks of fainting.

On August 17th, while at work, was suddenly seized with severe pains in epigastrium and faintness. For several days previously had been constipated, but on the 17th his bowels acted and he felt relieved. On August 19th, while at work, the pain and faintness recurred, whereupon he was sent to the hospital.

On admission he was semi-collapsed, and complained of severe epigastric pain. Is extremely pale and cold. Temperature 96·4°. No marked thoracic or abdominal physical signs were made out. After treatment he slowly recovered from his collapse, but early next morning he became suddenly worse, vomited a pint of blood, became comatose, and died an hour later.

Post-mortem examination (see P. M. Book, vol. vi. p. 357) was made twenty-nine hours after death.

Body well nourished. Blood issuing from nose and mouth. Abdomen distended. Gaseous distension of subcutaneous and submucous cellular tissue in various parts. P. M. decomposition well marked. Varicose veins of lower extremities. Brown pigmentation of skin of both legs. An ulcer about the size of a shilling just above right internal malleolus. Costal cartilages ossified. Chest barrel-shaped. Pleuræ smooth and shiny. The lungs are well-marked examples of hypertrophous emphysema. There are some few fibrous nodules immediately beneath the pleuræ. Bronchitis.

Pericardium normal. Heart 18½ oz. Valves competent. Endocardium normal. Aorta normal.

Fauces normal. Stomach very large and distended with about two pints of black clot and blood. The first part of the duodenum is dilated, resembling a small second stomach. It is the seat of a punched-out ulcer about the size of a florin. Its base is partly formed by the pancreas. In the floor is seen a longitudinal ulcerated slit about ¼ inch long. This opens into a large artery, the pancreatico duodenalis.

The œsophagus and intestines, as well as the stomach, contain fluid blood. The gastro-intestinal mucosa is of a dark red colour from being stained with the dissolved out hæmoglobin; with the exception of this staining and of the ulcer it is not noteworthy.

Spleen (7 oz.) very soft, spongy, and discoloured. Liver (71 oz.) soft, discoloured, and flabby. Kidneys 4¼ oz. and 4¾ oz. Capsules thick and adherent, cortices wasted, surfaces granular, consistence firm, colour pale.

Numerous bosses of bone along the bodies of the vertebræ; joints of hands and feet large.

No evidence of urate of soda in auricles, but there is a copious deposit of these crystals in all the joints, except the hip and shoulder joints. In the smaller joints the cartilage is eroded. Brain: Dura thick and adherent. Vessels of pia mater normal. In the right basal ganglia, but chiefly in corpus striatum, are several small cavities with sharply defined margins. There is no thickening of their edges, nor are there any appearances of inflammation or degeneration round about them.

Microscopical examination of the cartilage of the knee-joint: vertical sections show continuous deposit of urate of soda crystals very near to, but not quite reaching, the surface of joint. Towards the bone the cartilage is quite free from urate. The deposit is irregular in quantity, being thicker in some places than in others.

There are, too, spheroidal aggregations of these crystals, which impart the notion that they may be in cartilage cells. If so, it is a matter of accident, for the sections generally show that the crystals have no special predilection for the cells. At any rate they do not present the artistic appearance depicted in the illustration of Cornil and Ranvier.

REMARKS.

Two cases with a greater number of contrasts than those just described would be difficult to find. All that they have in common is the site of the lesion and the "punched out" character of the ulcer. Perhaps it would be convenient to state that I am here speaking of that ulceration of the duodenum which is described under the name of simple, perforating, chronic, duodenal, an ulceration having many features in common with a similar lesion more frequently seen in the stomach.

In both the cases there was a lesion of the skin, but in neither instance can it be properly maintained that there was any real connection between the cutaneous sores and the intestinal ulcers. For in the first instance the skin lesion, which was quite superficial, did not appear till after the abdominal symptoms were well marked, and in the second the supposition that the varicose ulcer was causatively connected with the duodenal ulcer, would naturally suggest a more frequent occurrence of duodenal and gastric ulceration. Of course, I do not mean to assert that there is any sheer impossibility in the connection of a varicose ulcer of the leg and an ulcer of the duodenum; indeed, my belief is that, as in religion so in medicine, all things are possible.

The ages of the patients are in marked contrast. In one it is three months; in the other it is sixty-three years. The one is a suckling infant fed on the breast only; the other is a gouty old man, who in all probability had been endowed with that voracity for which gouty people, at any rate for some time in their lives, are celebrated.

In the early period of life the chances of duodenal ulceration are, arguing from the recorded cases, quite small, while after sixty years of age the liability is very nearly, though not quite, eliminated.

Though the age of the first case was but three months, I find that a still earlier instance of ulcer of the duodenum is recorded (see Medical Press and Circular, 1878, p. 88: Woods). In this instance it was congenital. Soon after the infant was born it began to vomit blood, for which it was taken to the doctor. Death took place thirty-five hours after birth. The post-mortem examination revealed pneumonia of the right lower lobe, peritonitis, and a circular ulcer 1 cm. in diameter on the posterior wall of the duodenum. Both parents repelled the accusation of syphilis.

This and the one I have related are the only two cases in infants I know of, and in children only three are recorded, but two of them were consecutive to burns.

From statistics collected from various sources, and including all kinds of cases, it would appear that the period of greatest liability is in the third decade.

With regard to the pathology of duodenal ulcer, all sorts of speculations have been made. The most recent hypothesis, founded on experiments on animals, is that duodenitis may be set up by excretion of certain poisons through the bile. Other hypotheses are lesion of the mucosa from the irritation of some foreign body, thrombosis or embolism leading to necrosis of the mucosa. The necrosis is the necessary precursor of the solution by the gastric juice, the acidity of which is said to be much increased.

I have enumerated these items in order to see if the morbid appearances found in Case 1 could be illuminated.

In this case there were a perforating ulcer, an ulcer of mucosa, and a hæmorrhage into mucosa, all close to pylorus. The biliary papilla and its vicinity were quite normal.

The liver and the bile may, therefore, be excluded from having participated in producing the ulcer.

Now, is the hæmorrhage into the duodenal mucosa to be considered as forming the first, or preliminary, stage of the ulceration? That is not only possible, and even probable, but I don't go farther than that. The reaction of the contents of the small intestine were certainly strongly acid.

There was nothing to support a supposition of erosion by a foreign body, considered as a starting point of ulceration, nor any evidence in favour of thrombosis or embolism. The only tangible fact left out of which something may be made is that of the hæmorrhage into the mucosa.

In order to see if any light could be thrown on this question I looked over the P. M. Reports of Westminster Hospital for the past seven years. I find therein eight recorded cases of ulceration of the duodenum. In six of these death resulted from perforation. One of them was extremely interesting. A male, age thirty-one years, was admitted under Dr. Sturges on October 2nd, 1884, and died next day from peritonitis. At the post-mortem examination a large perforating ulcer of the duodenum was found. But there was also very advanced phthisis to which, had he not been carried off by the ulcer, he must have succumbed very shortly.

Microscopical examination of the lungs showed the presence of large numbers of tubercle bacilli, some of which were found in the walls of the pulmonary arteries.

Of course this is not much by itself, but it becomes significant by attaching to it the account of a gastric ulcer in an adult male, who died of hæmorrhage from this ulcer. This patient was admitted for phthisis, and at the post-mortem examination a large gastric ulcer and advanced phthisis were found. Microscopical examination of the ulcer showed the presence of giant cells within its wall, but I did not find any tubercle bacilli therein.

Another case bearing on the point is recorded by Dr. Murchison, in Path. Soc. Tr., 1869, vol. xx. pp. 174-6, where the author tries to show that the phthisis, of which the patient died, was the successor of the duodenal ulcer found at the post-mortem examination.

If the case of the infant be linked with the three cases just cited, the mere possibility is exalted into the probability that ulceration of the upper part of the gastro-intestinal tract may be dependent on the presence of microbes within the mucosal blood-vessels, or within the gland follicles, just as it is lower down, as in enteric fever, or even in tuberculosis.

From having looked over the results of many cultivation experiments with micro-organisms, experiments on the quality

and reaction of the nutrient medium, I am inclined to support the view which suggests some antecedent, or, at any rate, very early, alteration in the acidity of the gastric secretion. Indeed, a supposition of this nature is almost necessary for maintaining an ulcer of the gastro-intestinal tract as an ulcer, otherwise the tendency would probably always be to heal.

And the nature of the chemical reaction of the intestinal juice has, so I have often thought, something to say to the settlement of micro-organisms in certain special localities, for example, the lower end of the ileum.

With regard to the second case, I can only say that its appearance warranted the supposition that it had existed for quite a long time. Whether it had (previous to a month before the patient's death) caused any inconvenience, or was productive of any important symptoms, it was impossible, from the condition of the patient, to ascertain. At any rate, the case was all round extremely interesting and instructive.

I have never yet been able to verify the connection between burns and ulceration of the duodenum. The Westminster Hospital Reports contain records of only six cases of burn from the year 1880 up to the present time. In none of these was there any trace of inflammation in any part of the gastro-intestinal tract, but in a case of scald it is noted that the vessels of the duodenum and the first part of colon were injected.

NOTE ON THE ACTION OF APOMORPHINE AS AN EXPECTORANT.

By Dr. MURRELL.

Apomorphine is steadily gaining ground as an expectorant. A good deal of misconception still exists respecting the action and properties of this valuable drug. The popular idea is that it is chiefly useful as an emetic, and that it should always be given hypodermically. This is an entire mistake. The chief use of apomorphine is as an expectorant, and when employed for this purpose it should be given by mouth. It is not intended for one moment to deny that it is an emetic, for it is undoubtedly the most valuable emetic we possess; but the demand for emetics is small, and half a dozen emetics will last a man a lifetime. Expectorants, however, are on an entirely different footing. Most of us who live in London suffer during the winter months, more or less from bronchitis, or at all events bronchial catarrh, and the great bulk of the patients on the medical side of the out-patient department seek relief for chest affections. For them expectorants are a necessity, and they consume them in almost unlimited quantities. When a man comes to the hospital in a state of intoxication, he obtains relief from a single dose of an emetic, but the chronic bronchitic comes week after week and month after month, and is by no means an easy person to get rid of. For every dose of apomorphine given to induce emesis, hundreds are given to promote expectoration.

This is only one preparation of apomorphine in the British Pharmacopœia, a 1 in 50 solution of the hydrochlorate, and this rejoices in the misleading title of "Injectio apomorphinæ hypodermica." It is not only that the name is misleading, but the strength is inconvenient for dispensing purposes. It must be remembered that it was introduced and made officinal

as far back as 1885, when the real use and properties of apomorphine were only just beginning to be recognised. In the Pharmacopœia we are told that the solution should be prepared as required for use, and this is another mistake. It is quite true that the solution turns green if kept for more than a few hours, but this change of colour in no way interferes with its activity either as an emetic or as an expectorant.

A good deal of misconception exists, too, as to the dose in which apomorphine should be administered. It may be given by mouth quite freely and several times a day without fear of exciting nausea or vomiting. A drachm of the officinal solution is certainly not too much, and I have often given a grain of the hydrochlorate four times a day without producing the slightest discomfort. The only precaution I take is to see that it is given after meals. I generally give it in a mixture or linctus with a few drops of dilute nitro-hydrochloric acid—which gives it a red colour—and flavour it with glycerine or syrup of tar or syrup of Virginian prune, or any other flavouring agent which may seem to be especially indicated. It is not allied in any way in action to morphine, and a combination of acetate of morphine and hydrochlorate of apomorphine often proves extremely useful in the treatment both of phthisis and chronic bronchitis. Children take apomorphine well, and when mixed with vaseline or lanoline and rubbed into the abdomen it greatly facilitates expectoration. Of late I have used it in the form of a spray with considerable benefit. Apocodeine has much the same action as apomorphine, but it is not a commercial product, and its present high price is a bar to its general use as a therapeutic agent. For a detailed account of the history and uses of these drugs see my paper "On the Action of Apomorphine and Apocodeine, with reference to their value as Expectorants in the Treatment of Chronic Bronchitis," in the *British Medical Journal* of February 28th, 1891.

THREE MAJOR AMPUTATIONS.

UNDER THE CARE OF MR. COWELL.

REPORTED BY MR. GREEN.

Dislocation of the right radius forwards; fracture of the right ulna; rupture of the interosseous artery; traumatic aneurism; primary amputation of the arm; union by first intention.

HENRY CHARLES P—, æt. 37, a carman, was admitted on October 19th into Northumberland Ward under Mr. Cowell. At 9.45 a.m. the patient was in charge of a horse and cart, when the horse took fright and knocked him down, the wheel of the cart going over his right arm just below the bend of the elbow. He came to the surgery about twenty minutes after the accident with a great amount of swelling, due to effusion of blood in front of the right elbow-joint, obliterating the natural folds in the skin; slight pulsation could be felt over the swelling, which fluctuated distinctly. The right radius was dislocated forwards, and the ulna fractured at the junction of the upper with the middle third. No pulsation could be detected in either the radial or ulnar artery at the wrist. Fingers and forearm blue and cold, and skin tightly stretched threatening to burst. The limb was bandaged, a tourniquet applied to the brachial artery, and Mr. Cowell was immediately sent for. At noon the patient was put under ether, the skin antisepticised, and the arm amputated in its middle third, by two square flaps anterior and posterior of equal length. The humerus was sawn in its middle, the brachial artery tied with a carbolised silk ligature, and a few muscular branches secured with carbolised catgut ligatures before the tourniquet was removed.

A large amount of blood-clot and fluid blood was turned out, the muscular planes being extensively infiltrated with blood.

There was a very slight amount of hæmorrhage during the operation.

The flaps were brought accurately together with horse-hair sutures, and two small drainage tubes inserted in either angle of the wound. A carbolised bandage was applied to steady the muscles. Wound powdered with iodoform and dressed with pads of double cyanide gauze, iodoform and sal-alembroth wools, and the whole bandaged firmly to a splint. 12 p.m., dressed. Sanguineous discharge has come through the dressings. Temperature 100° F. Patient feels quite comfortable.

October 20th, 11 a.m. Dressed; both drainage tubes shortened.

October 26th. Dressed; both drainage tubes removed; no rise of temperature since the first dressing; the temperature is now normal.

November 2nd. Dressed; the wound has almost healed; there is a very slight amount of discharge.

November 6th. Dressed; wound soundly healed; all the sutures removed; was up for 2 hours yesterday; temperature has never been above normal since the last note.

November 19th. Discharged. Is going to a convalescent home.

Examination of the limb after removal.—The radius is dislocated forwards. The ulna is fractured transversely just below the insertion of the brachialis anticus, the upper fragment being flexed by this muscle; the lower fragment is drawn upwards and inwards, the interosseous artery being torn across by its jagged upper end, close to its origin from the ulnar artery. There is hæmorrhagic effusion into the elbow joint, and the tissues are extensively infiltrated with blood. There is no fracture of the humerus.

Remarks by Mr. Cowell.—In this case neither the fracture nor the dislocation were compound, but the joint was evidently lacerated. The extensive extravasation into the limb, in addition to the other injuries, left no option as to the propriety of immediate amputation. The rapidity with which the stump healed is unusual in primary amputations.

Strumous disease of the left knee-joint; amputation of the thigh; slight secondary hæmorrhage. Recovery.

Mary L—, æt. 35, housewife, was admitted on October 7th into Hollond Ward under Mr. Cowell.

Ten years ago, following a confinement, the patient was laid up with a "white leg." The swelling of the left thigh and leg disappeared in about 6 weeks, the left knee-joint however still remaining enlarged. During the last 2 years the swelling of the joint has increased considerably in size, and for the past 6 months the patient has had starting pains in it at night. The patient experiences a feeling of weakness and insecurity about the joint on walking, but no actual pain. Has lost flesh. Never had hæmoptysis. No history of phthisis in the family.

The left knee-joint is much enlarged, being about twice the size of the right. The swelling is partly solid, partly fluid. Distinct fluctuation can be felt in the joint. The ends of the femur and tibia are much enlarged, On the inner side of the patella, which is hidden in the swelling, is a red and tender spot.

October 8th. Mr. Cowell made two incisions into the joint on either side of the quadriceps extensor tendon, in order to drain the joint. A small amount of clear serous fluid and gelatinous material escaped, the size of the joint being only very slightly diminished by the operation. The cavity of the joint was syringed out with carbolic lotion 1 in 40, and dressed with double cyanide gauze, iodoform, and sal-alembroth wools.

October 11th. Since admission the temperature has not risen above 101° F., but at 4 p.m. to-day the temperature rose to 104° F. Respirations 18. Pulse 108.

October 13th. Temperature 104·6° F. The patient being under ether, Mr. Cowell enlarged the previous incisions. The pulpy mass in the joint was very dense and firm, and it was only possible to scrape away a small quantity of gelatinous matter. Two drainage tubes were inserted, and the wounds were dressed and syringed as before.

October 15th, 4 p.m. Temperature 104° F. As the patient is evidently beginning to suffer from the continued fever, Mr. Cowell decided to remove the disease by amputating through the lower third of the thigh. The patient being under ether, the skin antisepticised, and a tourniquet applied to the

femoral artery, a long anterior flap, composed at first of skin and subcutaneous tissue only dissected carefully from the caseous structures of the degenerating joint, and then of the whole thickness of the muscles down to the bone, was made by cutting from without inwards, and a shorter posterior flap fashioned in the same manner. The femur was sawn rather below the centre of its middle third. The femoral artery and vein were secured with silk ligatures, and several other smaller muscular branches with catgut ligatures, before the tourniquet was removed. There was very slight hæmorrhage. The flaps came into accurate apposition, and were sutured together with silk, making a good square stump. A drainage tube was inserted from one side of the wound to the other. A carbolised bandage was applied round the thigh to steady the muscles, iodoform powder dusted over the wound, pads of double cyanide gauze applied, and the whole thickly covered with iodoform and sal-alembroth wools, and bandaged to a back splint. At 8 p.m. the temperature fell to normal, but rose to 100·2° at 12 p.m.

October 16th, 12 a.m. Temperature 103·4° F. Dressed; drainage tube divided and both pieces shortened; wound healthy; dressings stained with sero-sanguineous discharge.

October 17th, 8 p.m. Temperature 103·2° F. Dresssed; flaps distended with blood-clot, no doubt due to some small vessel having bled on the patient's recovering from the shock of the operation; several stitches and blood-clots were removed, and the wound was syringed out with carbolic lotion 1 in 40; the drainage tubes were also removed.

October 18th, 4 a.m. Dressed; temperature 101·6° F.; wound perfectly sweet; discharge sero-purulent and free; put on 6 ozs. of brandy.

October 30th. Since the 18th, the stump has been syringed out with carbolic lotion and dressed twice daily, iodoform powder being dusted into the wound. The wound is now granulating healthily from the bottom, and discharges a small quantity of laudable pus. The outer angle of the stump has united by first intention, but on the inner side there is a cavity fully 3 inches in depth. The temperature now remains about 100° F., and the patient eats, drinks, sleeps, and feels well.

November 3rd. Temperature 99° F. The wound has been dressed once daily since the 30th, is granulating healthily, and

much smaller. There is about an ounce and a half of perfectly sweet pus with each dressing. Has no pain in the stump.

November 7th. Dressed last on November 5th, when the flaps were bandaged firmly together with a carbolised roller. This morning they have apparently united by "secondary adhesion" of the two healthy granulating surfaces. There is only slight purulent discharge from a small opening in the centre of the end of the stump, and on the inner side where the drainage tube was. Brandy changed to 4 ozs. of port wine.

November 9th. Dressed; the flaps are firmly united; there is hardly any discharge. The patient has had some diarrhœa since October 31st; this has now ceased, the bowels acting twice daily.

November 14th. Dressed; last dressing on November 9th; the stump looks now as if it had healed by first intention.

November 20th. Dressed; only a few drops of pus from the inner angle of the stump; was up for 3 hours to-day.

November 26th. Dressed; no discharge; wound healed soundly; gets up every day; scar well behind the bone; no tenderness about the stump.

December 1st. Is still in hospital, but only waiting to go to a convalescent home. She has gained flesh and is much improved in appearance.

On laying open the joint the synovial membrane, as well as the soft structures around the joint, were found to be much thickened. There was a large mass of degenerating tissue in the joint, and the synovial fringes formed grape-like bodies, varying much in size. It was some of these that were removed through the incisions. The cartilages were thin in places, but remarkably sound when the amount of pulpy growth and the duration of the disease are considered.

The stump is of good square shape, a point on which Mr. Cowell lays much stress.

Acute necrosis of the tibia; amputation of the thigh; septic pneumonia. Death.

William B—, æt. 15, errand boy, was admitted on November 4th into St. Matthew Ward, under Mr. Cowell. Two months ago patient began to have pain in the left great toe, due to the nail

growing-in and pressing against the skin. The nail looks yellowish white and sodden, and is partially separated from its bed; the grooves on either side are ulcerated and sloughy, and discharge ichorous fetid pus. The patient complains of pain and tenderness up the inner side of foot to just above the ankle. There is some swelling about the ankle, especially on the inner side, and a few lines of superficial redness, corresponding to the lymphatic vessels, can be traced running up from the base of the great toe to just above the ankle. Temperature at 12 a.m. 99·2° F., at 7 p.m. it rose to 104° F.; 7.45 p.m. patient put under gas, the toenail removed, and boracic fomentations applied, has great tenderness on pressure over the lower third of tibia; 8 p.m., quininæ sulph. grs. vi.

November 5th, 3 a.m. Temperature 102·4° F.; quininæ sulph. grs. v; belladonna fomentations to be applied to the leg every 4 hours. Temperature at 11 p.m. 103·6° F. The bowels have acted 6 times during the day. Urine sp. gr. 1035 acid, no albumen, phosphates, no sugar.

November 6th, 3 a.m. Temperature 102·6° F.; quininæ sulph. grs. v. The leg is much more swollen to-day, especially about the ankle, on the outer side of which fluctuation (?) can be felt. The patient does not feel any pain unless he moves the limb. Temperature at 11 p.m. 103·6° F.

November 7th, 12 a.m. Temperature 103° F. Distinct fluctuation can be felt over the lower end of the tibia. Patient put under ether, and two incisions, each 3 inches in length, made over the front of the tibia, through the periosteum, over the middle and lower thirds of the bone. A large quantity of thick, fetid, unhealthy pus escaped. On examining with the finger, the periosteum is stripped up from the whole of the anterior surface of the shaft of the tibia, the bone being laid bare. The cavity was washed out with carbolic lotion 1 in 40, a drainage tube inserted, and the wounds dressed with strips of double cyanide gauze, iodoform, and sal-alembroth wools. 3 p.m. temperature fell to 101·2° F. Wound to be syringed out with boracic lotion, and boracic fomentations to be applied every 4 hours. 11 p.m., temperature 103·2° F.

November 8th. Put on 4 ozs. of port wine.

November 9th, 2.30 p.m. Patient put under ether, and the previous incisions enlarged by Mr.

November 10th. The two incisions laid into one, making one large wound over the front of tibia the whole length of the diaphysis. The skin just below the inner side of the knee-joint is red, œdematous, and very tender on pressure. The patient cries out with pain whenever the limb is moved to renew the fomentations. Temperature 104° F.; pulse 132; respirations 24. Has a slight dry cough; does not complain of pain in any of the other joints; tongue very furred; takes fluid nourishment well; bowels open daily. 8.30 p.m., Mr. Cowell saw the patient. The redness, tenderness, and œdema noticed this morning have extended up the inner half of the thigh. The skin is to be painted over with a solution of nitrate of silver grs. xx, ad. ℥i every 4 hours.

November 11th. Patient slept well during the night. Temperature at 11 a.m. 103·2° F.; respirations 21; pulse 120. Tongue furred, but cleaner than it was; still has a slight dry cough. Looks a little better.

November 12th. Temperature at 3 p.m. 104·6° F. Mr. Cowell ordered antipyrin grs. xv, and quininæ sulph. grs. v alternately every 4 hours. Limb put on a double-inclined plane, in order to prevent the tendency which the discharge has to gravitate towards the knee. There is not so much redness or swelling about the knee-joint as there was yesterday. A large quantity of purulent discharge comes away with each fomentation.

November 15th. At 8 p.m. temperature rose to 104·4° F. The knee-joint is much more swollen and tender; fluctuation can be distinctly felt in the joint; the lower third of the femur is tender on pressure, the patient wincing on its being handled. The patient looks much worse. The face and lips are dusky; pulse 150, feeble and very dicrotic; respirations 24. 11.45 p.m., Mr. Cowell came and decided to amputate the limb at once. The patient being under ether, the skin antisepticised, and tourniquet applied to the femoral artery, Mr. Cowell amputated the limb in the lower third of the thigh by a long anterior square flap, with the corners slightly rounded off, fashioned by cutting from without inwards, and a shorter posterior flap, cut by transfixion. The femur was sawn about the junction of the middle with the lower third, the cut section being quite healthy, and there being no pus under the periosteum. The artery was picked up and tied with a stout carbolised silk ligature, the vein

being secured with a carbolised catgut ligature. On relaxing the tourniquet there was very slight hæmorrhage, owing no doubt to the feeble action of the heart, several small muscular branches were picked up and tied with carbolised catgut ligatures. The flaps were secured accurately in apposition with carbolised silk sutures. Two drainage tubes inserted in either angle of the stump, the wound dusted over with iodoform crystals, a carbolised bandage applied firmly round the stump to steady the muscles, pads of double cyanide gauze, iodoform and sal-alembroth wools applied, and the stump bandaged to a back splint. A hypodermic injection of morphia, 3 minims, was given before the patient left the operating theatre. The pulse and aspect of the patient improved immediately after the amputation was complete.

November 16th, 11 a.m. Temperature 99·4° F. The pulse has improved in quality and is not so frequent. The patient slept well last night after the injection, and looks much better. 2.15 p.m. Dressed. The dressings are saturated with a large amount of sero-sanguineous discharge; syringed out the wound with carbolic lotion 1 in 40. 5.45 p.m., dressed as before, with the exception of syringing, as the discharge has come through the dressings. 11 p.m., temperature 102·6° F. Inj. morph. hypodermica, ♏ iij.

November 17th, 11 a.m. Slept well last night after the injection. Temperature 103·2° F. Dressed; wound syringed out with carbolic lotion; both drainage tubes shortened; discharge sero-purulent and free. Ol. ricini ℥ss. statim; the bowels have not acted since the 14th. Port wine increased from 4 to 6 ozs. Pulse 132; respirations 27. Has a cough, with slight expectoration; has slight præcordial pain. Examination of front of chest and heart nil. Bases not examined, as patient cannot be sat up or rolled over. Has no joint pains. Face and lips dusky. Tongue coated with a moist white fur. Does not take fluid nourishment so well this morning. Urine acid, sp. gr. 1022, no albumen, no sugar. ℞. Quin. sulph. grs. iv, in pill every 4 hours.

November 18th. Slept fairly well during the night, very delirious during the intervals when awake. Has frequent cough with small amount of rusty-coloured expectoration. Has no pain in the stump or any other part of the body, with the excep-

tion of that over the xiphoid cartilage on coughing. Takes nourishment better to-day. The bowels acted freely after the castor oil. Pulse 129, regular; respirations 33; temperature 11 a.m. 103·2° F. Is unable to take the pills; the same quantity of quinine in powder to be given every 4 hours. The tongue is cleaner; 6 ozs. of brandy to be given instead of the port wine. 5.30 p.m., dressed; large amount of serous discharge. Is very delirious to-day, but answers rationally when spoken to. Passes his urine and fæces unconsciously into the bed. Pulse 132, regular; respirations 36. Urine acid, sp. gr. 1025, no albumen, no sugar.

November 19th, 11 a.m. Temperature 101·6° F.; pulse 138; respiration 36. Has had more cough and expectoration during the night; is still very delirious. Fine crepitations can be heard in both axillæ, more marked in the right. Unable to examine the back, as patient is too ill to be raised. Still has pain on coughing over the xiphoid cartilage. Sputum rusty-coloured and muco-purulent. 2.30 p.m., Mr. Cowell saw patient. Dressed. No appearance of union between the flaps. On syringing the lotion comes through between the stitches. Is very drowsy and delirious this afternoon. Still passes his urine and fæces unconsciously into the bed. 3 p.m., temperature 103·8° F. Cough and expectoration as before. No swellings to be detected in any of the joints, or other parts of the body. Breath smells sweetish. Respirations 42; pulse 132. The bowels have acted twice. Put on bark and ammonia instead of quinine.

November 20th. Temperature 7 a.m., 104·6° F. 4.30 p.m., dressed. The flaps are simply held together by the sutures; drainage tubes removed; urine acid, sp. gr. 1025, no albumen, no sugar. Temperature 11 p.m., 102·8° F.; respirations 54; pulse 160.

November 21st. Patient much weaker, delirious and unconscious. Temperature 11 p.m., 105·4° F.

November 22nd. Died at 11.30 a.m. Temperature just before death 107° F. A post-mortem examination was refused.

Examination of the limb after removal.—The periosteum is everywhere separated from the shaft of the tibia. On opening the knee-joint it is found to be full of pus. The skin of the dorsum of the foot is œdematous from serous effusion into the subcutaneous tissues, and substance of the skin. There is no

purulent effusion into the ankle-joint. The lower epiphysis of the tibia is completely separated from the diaphysis.

There is no clue in this case to the source of the infection. There was no wound but that caused by the in-growing toe-nail, and it is probable that that wound was the portal through which the materies morbi found an entrance. The high temperature and other symptoms had commenced before the operation of removal of the toe-nail was performed.

THREE FATAL CASES OF ENTERIC FEVER.

REPORTED BY MR. SHARMAN.

THE following three consecutive cases were admitted into the medical wards during September, 1891. The first on September 4th, the second on September 7th, and the third on September 25th. All three cases occurred in young adults, their ages being respectively 17, 19, and 21. Two of the patients were males, one a female. They all terminated fatally.

Cases 1 and 2 were of somewhat similar character, and ran about the same course. Both seem to have contracted a severe form of typhoid, and to have died from the severity of the attack, or of septicæmia, at the 11th and 15th day respectively after the onset. Both patients had great prostration and mental disturbance, beyond which there was no complication, except hyperpyrexia, which was a marked feature of the end of both cases.

In Case 1 the temperature had a high range throughout, for although kept down to some extent by cold sponging, it remained the greater part of the time between 104° and 105°, and on the day of his death rose to 106·6°. The pulse, except on the last day, never exceeded 108.

Case 2, although very prostrate and at times taking her food badly, seemed to run a fairly favourable course before the temperature rose suddenly to 108·8°, death occurring one hour later, the temperature then being 109·4°.

There seems some doubt as to whether this patient contracted the disease in Cardiff or London. If in Cardiff, it would give an unusually long incubation period of at least 25 days, as she left that town on August 10th, and the day of onset seems to have been September 3rd. In favour of the supposition that she contracted the disease in Cardiff is the fact that her sister, who

occupied the same bedroom, was taken ill with enteric fever on August 27th; she has since recovered. The Medical Officer of Health, Dr. Walford, kindly informs me that the sanitary arrangements of their house were in good order, but that at the business premises, where the patient worked, the drains were in a defective condition.

Case 3 on admission looked extremely ill. He had frequent attacks of diarrhœa, which ceased for about 24 hours after a rectal injection of starch and opium. Except for this diarrhœa, his attack seemed to run a comparatively mild course up to the 23rd day of disease, when symptoms of perforation suddenly set in, and he died about 24 hours after.

Case 1.—J. S.—male, æt. 17, a porter, was admitted on September 4th into Hallett Ward, under Dr. Sturges. He stated that he had suffered from severe frontal headache, pains in the back and limbs, and a general feeling of illness for a fortnight. He had not been to work on account of his illness for 14 days, and had spent most of that time in bed. He had also suffered from anorexia and thirst. His bowels had been open twice daily, but there had been no loose motions. His mother said that he had been much worse since August 31st, and that he had been very delirious during the night of September 3rd.

Some drains were being repaired in the vicinity of the house; there was, however, no bad smell from them, and no one else was ill in the same house. He had had no previous serious illness. Family history was unimportant.

On admission (September 4th, probably about the sixth day of disease) the temperature was 102°, rising in the next four hours to 105·2°. The pulse was 100, regular, of small volume, and compressible. Respirations 24 to the minute. Urine was acid, 1020. Albumen (a cloud). His face was flushed, he looked ill, and was breathing heavily. The tongue, mouth, and throat were very dry. He was very drowsy and slightly delirious. The abdomen was distended, there was no distinct localised tenderness. The area of splenic dulness was enlarged, but the spleen could not be felt. There were numerous rose-coloured spots, slightly raised, and disappearing momentarily on pressure, on the back and abdomen.

A few hours after admission he became more delirious, and

tried to get out of bed. He was ordered to have quinine sulphate, grs. 5; was sponged four times in the 24 hours with cold water, reducing the temperature slightly on each occasion.

September 5th, temperature continued between 104° and 105°, pulse 104, respirations 38. He passed a large yellow liquid motion in the bed. Tongue and mouth dry and brown; some fresh spots were noticed. Was given 5 grs. of quinine, and sponged twice.

September 6th, abdomen was more distended, and there was some tenderness over the spleen. Temperature 102° to 104°. Delirium had continued without intermission since admission.

September 7th and 8th, condition remained about the same. Each day he passed two light liquid motions in the bed. On the 7th was ordered to have 2 ozs. of brandy in the 24 hours; this was increased to 6 ozs. on the 8th.

September 9th (11th day of disease). Temperature at 3 a.m. was 104°. He had had a very restless night, and at 5 a.m. the temperature rose to 106·2°, and 106·6° at 6.30 a.m. Ice bags were applied to his head, axillæ, and groins, and lumps of ice were placed along his body, reducing the temperature to 103° by 8 a.m. The pulse had increased in frequency from 104 at 4.30 a.m., to 132 at 10 a.m. The tongue was dry and brown, the mouth ulcerated. He passed motions and urine beneath him unconsciously. The urine contained more albumen. The temperature was controlled throughout the day by means of ice, but his condition continued to grow worse, and he died at midnight, the temperature at death being 106°, the ice having been left off two hours previously on account of the cyanosed condition of the patient.

A post-mortem examination was made by Dr. Hebb 14 hours after death. The body was well nourished; post-mortem rigidity well marked, and there was slight lividity of surface of body. The intestinal glands in the lower half of the ileum were greatly swollen, somewhat firm, and were standing well above the level of the mucosa. Towards the ileo-cæcal valve there was a good deal of ulceration, but this was a less marked feature than the swelling of the solitary and agminate glands. The gut was filled with liquid bright yellow fæces. The stomach was small and the walls thin. Peritoneum normal. Abdominal lymphatic glands were quite large, soft, and hyperæmic. The

spleen weighed 17 ozs., was firm in consistence, and of a dark purple colour. Liver 58 ozs., pale. Kidneys weighed 6 and 5¾ ozs., pale and flabby. There were a few old adhesions over both lungs, and excess of fluid in both pleural sacs. The posterior halves (with exception of the extreme bases) of both lungs were consolidated, blackish, friable, and œdematous. There was some bronchitis. The mucosa of the posterior surface of the epiglottis was ulcerated. Pericardium, heart, and bladder normal.

Case 2.—M. M.—female, æt. 19, single. Admitted into the West Extra Wards on September 7th, 1891. This patient left her home at Cardiff on August 10th in good health, except that she had lupus, affecting the nose, for which she had been treated by Dr. Fox on and off during the last five years. She was admitted into Hollond Ward on August 12th, under Mr. Stonham, who scraped her nose. She remained in the Surgical Ward for 10 days. During that time her temperature was normal, except for an occasional evening rise to 99° and 100°. After leaving Hollond Ward she lived with relatives in Clapham. She was brought to the hospital again, 14 days after her discharge from the Surgical Ward, and stated that she had not felt in good health since her operation 4 weeks before, having suffered from headache, and felt tired and languid. On September 3rd she had a sore throat, had anorexia and nausea, and felt very weak. On September 7th (day of admission into the Medical Wards) she complained of slight pain and tenderness in abdomen, had pain in all her limbs, and was sick once; she had had constipation during the past 4 weeks, and her bowels had not been opened last 4 days. Family history was good. A sister who occupied the same bedroom as the patient at Cardiff had been ill since August 22nd, and in bed since August 27th, with enteric fever.

On admission her temperature was 103·6°, pulse 104, full volume, and regular. The tongue was covered in the centre with white fur, clean at the tip and edges. The fauces were red and injected. The respirations were 24, respiratory movement was chiefly thoracic; there were a few moist sounds heard at the end of inspiration over the right back.

The abdomen was rather tumid, and there was general tenderness. There were several small vesicular spots on chest

and back, and four larger rose-coloured spots on abdomen, fading on pressure.

September 9th. The patient was rather drowsy; the temperature ranged between 104° and 101°, pulse 104, respirations 24. A simple enema (1 pint) was given, and acted well; the motion was light coloured and semi-liquid.

September 10th and 11th. There was some diarrhœa; she was very drowsy, and had tremor of the hands; tongue furred, dry, and brown.

12th. Was very delirious during the night. Ordered 2 ozs. of brandy in the 24 hours.

13th. Was much worse. Took her food badly. Passed urine and fæces in bed. Tongue very dry and brown, sordes on teeth; very delirious at night, tried to get out of bed. Brandy was increased to 4 ozs.

14th. Did not sleep during last 24 hours, and seemed much weaker. Pulse more frequent, being 124; respirations 44; temperature 102° to 103°. Ordered 8 ozs. brandy.

September 15th. Swallowed food with difficulty. Tremor and subsultus tendinum increased; several fresh enteric spots were noticed during the last three days. Bowels have been opened once or twice daily; motions loose and light.

September 16th. There was slight improvement. Ordered champagne, 1 pint, and 6 ozs. brandy.

September 17th (15th day of disease). The temperature rose to 105° at 6 a.m., after cold sponging came down to 102·6°; the apparent improvement of the previous day continued up to 6.30 p.m., the patient swallowing well, and answering questions fairly. After having some milk at 6.30, she had a rigor, and became cyanosed. The temperature rose rapidly to 108·8°; pulse was 136, very small volume. Lumps of ice were placed beneath and along the sides of the patient and over the large vessels. The temperature, however, continued to rise, reaching 109·6° just before death, which occurred at 7.30 p.m.

The post-mortem examination was made by Dr. Hebb, 19 hours after death. The body was well nourished. There was post-mortem lividity of the head and posterior surface of the body. Both anterior nares were much eroded by lupus, the nasal mucosa was covered with a thick yellow scab. All the internal organs and tissues were soft, flabby, and deeply stained

with hæmoglobin. The blood was frothy and fluid; larynx nil. There was slight excess of fluid in both pleural sacs; some hypostatic engorgement and bronchitis of both lungs. Peritoneum normal. The gastro-intestinal mucosa was swollen, soft, and hyperæmic, especially that of the last three feet of the ileum. In these three feet Peyer's patches were somewhat swollen, and near the ileo-cæcal valve there were some small patches of ulceration; in other respects the intestinal mucosa was not remarkable as a specimen of enteric fever. The mesenteric glands were large, soft, and hyperæmic. The spleen weighed 7½ ozs., was firm, brittle, and a dark red purple colour. Liver 53 ozs.; there were numerous old, thin, but tough peritoneal adhesions; the liver itself was a yellow drab colour, and very soft. There was nothing very noticeable about the other organs, except the ovaries, which were cystic.

CASE 3.—T. H., male, æt. 21, a laundry-man. Was admitted into King William Ward, under Dr. Sturges, on September 25th, 1891.

He gave the following history of his illness: On September 14th he had headache, and pains all over his body. About the same time he had fits of shivering, followed by sweating, which continued up to his admission into the hospital. For 7 days previous to admission his bowels had been open twice daily; the motions were loose and yellow. He had anorexia, no sickness, no cough, and no pain in abdomen.

He had had an abscess in the groin 14 years before, but no other serious illness.

September 26th. Temperature was 101° to 104°, pulse 108, regular, and good volume; respirations 28. His face was pinched, and he looked very ill. Tongue was dry, and coated in the centre with yellowish fur, clean at the tip and edges. He was slightly deaf. For the first time since the onset of his illness he complained of some pain and tenderness in the abdomen.

The abdomen moved slightly during respiration, was tumid; there was one rose-coloured spot noticed on abdomen. Spleen could not be felt.

The bowels were opened three times in the 24 hours; motions were of a light brown colour, and quite liquid. Chest normal.

September 27th. Did not sleep well on previous night on account of headache. The abdominal pain was continuous; sick twice; bowels open twice, motions liquid, light brown colour. Was given pulv. ipecac. co. grs. x.

September 28th. Face more pinched. Intellect was quite clear. Complained of great pain and tenderness in abdomen; several fresh enteric spots noticed. Was sick twice; had six watery, very light motions at short intervals. Was ordered an enema, containing tincture of opium, ♏ xx, and 2 ozs. of starch, which checked the diarrhœa for the next 24 hours.

September 29th. Some fresh spots seen on chest and abdomen.

September 30th. Temperature 101° to 103·8°. Headache was less, slept well. Was delirious occasionally through the night, but when roused answered questions rationally. Pulse 108, regular, soft, compressible, and dicrotic. The first sound of heart was more feeble; had a return of the diarrhœa, having eight loose motions during the day. The starch and opium enema was repeated with good effect. Ordered to have 2 ozs. of brandy in the 24 hours.

October 1st and 2nd. Was not so well. Tongue dryer, brown in centre, and tremulous; he was drowsier and more apathetic; the deafness had increased. Temperature was 102° to 103°, pulse 104, respirations 40.

October 3rd. A fresh crop of spots came out on abdomen; had seven loose motions, the last containing a few streaks of blood; the diarrhœa again stopped after a starch and opium enema. Was delirious through the night.

October 4th. Temperature 102° to 104°, pulse more frequent, being 112. Was very drowsy, and took his food badly; was sick once.

October 5th. Diarrhœa recommenced, but stopped after a starch and opium enema. Mind was quite clear, and he was reading a newspaper during the day.

October 6th. In the morning had six loose light motions in rapid succession; after the third he had a rigor, lasting 10 minutes, during which he was quite delirious. He later on complained of increased pain in abdomen. Pulse was 132, very small volume. By 4 p.m. the abdomen had become more distended; he was quite free from pain, he was sick after any

milk or stimulant, and passed two motions unconsciously in bed. Face very pinched; he had an anxious expression. Pulse 140. At 8 p.m. the temperature had fallen from 108° at 4 p.m. to 98°. The abdomen did not move during respiration. He was quite conscious through the night, and slept a little. The vomiting was continuous. He gradually became weaker, and died at 3 p.m. on October 7th.

In this case permission was given by the relatives for an examination of the abdominal cavity only. This was made by Dr. Hebb 44 hours after death. The body was spare. Abdomen was tense, and much distended, and on opening the peritoneal cavity much gas escaped. The peritoneal surface was injected, but except over a small patch superjacent to an ulceration of the ileum, there was no lymph or pus. The peritoneal sac contained several ounces of turbid bloody fluid. The gastro-intestinal canal was distended, partly with gas and partly with semi-fluid bright yellow fæces. The intestinal mucosa was ulcerated in the last three feet of the ileum; there was also some ulceration in the ascending colon. The ulceration was limited to Peyer's patches and the solitary glands; they were partly swollen and covered with yellow fæces, and partly in a condition of slough. In one ulcer there was a slough about the size of a pin's head; this slough had apparently not separated during life, but on passing some water through the gut it gave way. Before removal this ulcer was covered on its peritoneal surface with a thin layer of soft purulent lymph. There were in the ascending colon ten to twelve circular ulcerations, or sloughs, the largest not bigger than a threepenny piece. The mesenteric glands were large and soft. The spleen weighed 13 ozs., and was very soft. The kidneys were soft, pallid, and injected in places.

TWO CASES OF "HERPETIC FEVER."

REPORTED BY DR. GOSSAGE.

IN the second volume of the Westminster Hospital Reports Dr. Syers published five cases of pneumonia without physical signs of lung consolidation. These cases were most of them children, but one patient was an adult. In all the cases the onset was sudden, and accompanied by shivering; there was high fever, some dyspnœa, and during the course of the disease a herpetic eruption about the lips. The disease ended in all cases by crisis, accompanied by sweating.

The two following cases would seem to belong to the same class, although the dyspnœa was never so marked a symptom as in the instances recorded by Dr. Syers, and in one of the cases there was not a very definite crisis. One patient had some slight signs of lung consolidation, but these signs remained the same the whole time he was in the hospital. Pain in the sacral region was a prominent symptom with both patients during some period of their illness, but there was no particular complaint of pains in the limbs, as is most common in cases of influenza. These two cases possess a further interest, since they were brothers, and slept together; two other members of the same family were also affected by a similar complaint. The family consisted of a mother, a daughter aged 12, and three sons. The mother and daughter slept together in one room, the sons occupying another room. They dwelt in some workmen's dwellings in Westminster. There were no bad smells about the buildings, and there was no great prevalence of illness in them. The rooms inhabited by the family had been, however, recently painted and papered, and were damp when they went in to live there.

CASE 1.—J. G., male, aged 20, carman, was the first member to be attacked. He was quite well up to 10 p.m. on November 1st; he then shivered, and felt unwell. He had a slight cough, but no expectoration, and no pain in the chest. On November 2nd he was admitted into Burdett Ward, under Dr. Donkin, complaining of great pain across the sacral region, which he had first noticed that morning. On admission his temperature was found to be 102·2° F., pulse 108, and respirations 22 to the minute. The face was flushed, skin hot and dry; the patient was remarkably dull and prostrate; the tongue was moist, but thickly coated with whitish fur; the fauces were a little red and injected; the heart was normal. On examining the chest, it was found that there was a little less movement on the right side than on the left; vocal fremitus was increased over the lower part of the right back; a higher pitched note was obtained on percussion below the angle of the scapula on the right side; the vocal resonance was louder in this situation, and the breath sounds were not so loud when compared with the opposite side. Nothing abnormal found in the abdomen. There was some slight tenderness over the sacrum. At midnight the respirations had increased to 38 in a minute. During the night the patient perspired a little, but next morning he was more prostrate, and complained very much of the pain in the lower part of the back. The temperature was 102·2° F., pulse 96, respirations 18 to the minute. In the afternoon of this day, November 3rd, a herpetic eruption appeared about the lips, most marked on the left side. On November 4th the temperature was lower, and the patient better and brighter; much less pain in the sacral region. The herpetic eruption had by this time extended widely over the left side of the face, there being some even on the pinna of the ear. From this time the patient steadily improved, there being a slight nocturnal rise of temperature until November 7th, after which it remained permanently normal. The patient's convalescence was, however, rather protracted, and he was not judged fit to leave the hospital until November 18th. During the whole of his stay in the hospital the physical signs obtained over the base of the right lung remained the same, he being frequently and carefully examined, and they were the same the day he left the ward. He had no cough, and the respirations were only once found to be much accelerated—viz., on the first

night after admission. He stated that he had had inflammation of the lungs three years before, but was unable to say which side was affected.

On November 3rd the mother, Mrs. G., aged 49, was attacked suddenly with shivering, sore throat, and sore mouth. She had pains all over, especially in the back. On the third day after this initial rigor, an eruption appeared about the lips. She perspired on the evening of this day, and was much better the next morning, but sweated a good deal the next two nights. Mr. Thomas Bamford, of the Vauxhall Bridge Road, who attended this patient for a week, kindly informs me that she had a raised temperature, foul tongue, and constipation; the lips were covered with a herpetic eruption; there were no physical signs of disease in the chest, and there was no cough.

The mother informed me that her youngest son—P. G., aged 15—was attacked on November 7th with shivering, sore throat, pains in the back and head. He was feverish, and slightly delirious at night; he had no cough. On the third day of illness a slight eruption appeared on the face similar to the eruption the mother had. He sweated that night, was better next day, and quickly recovered.

Case 2.—S. G., male, aged 18, carman, was admitted in King William Ward, under Dr. Sturges, on November 8th. He had been taken suddenly ill on the morning of admission with shivering and pains in the head. He had had no previous serious illness. On admission the temperature was 103°, pulse 108, and respirations 32 to the minute. Patient was rather drowsy, the face was flushed, and the tongue furred. There was no cough, and there were no physical signs of disease in the heart, lungs, or abdomen. On November 10th the temperature fell to normal, without any marked sweating, and the patient felt quite well. On this, the third day of the disease, a crop of herpes appeared about the lips, most marked on the lower lip. From this time the patient steadily improved, and was discharged apparently quite well on November 18th. I am informed that he subsequently suffered a great deal from pain in the lower part of the back. He had never complained of a pain in this region while in the hospital. During his stay in the hospital there were never any physical signs of lung consolidation. About

3 weeks after being discharged this patient came to the hospital having been attacked with Herpes zona in the course of the lower dorsal nerves.

The sister of these patients, who slept with her mother, did not suffer from any illness about the time the rest of the family were ill.

FOUR CASES OF ACUTE INTESTINAL OBSTRUCTION, WITH REMARKS.

REPORTED BY MR. POWELL.

CASE 1. *Acute obstruction supervening on chronic obstruction, due to kinking and stenosis of the small intestine.*—Elizabeth D—, aged 52, was admitted into Hollond Ward on April 9th, 1891, with acute intestinal obstruction. Although complete obstruction had already lasted for 13 days, the patient did not look by any means alarmingly ill. The attack began suddenly on March 28th with severe pain in the upper part of the abdomen, quickly followed by vomiting. No cause could be assigned for the seizure, neither could any clue be obtained as to the probable nature of the occlusion. The woman had occasionally suffered from some slight pain in the epigastric region, and also from constipation, for the relief of which aperients were now and again taken; but in these respects she did not differ from the majority of women, and no notice was taken of the symptoms. The treatment, during the 13 days before admission, consisted in the administration of opiates and enemata, the latter bringing away some scybalous masses, but no flatus was passed. Vomiting continued at intervals during the whole time, and on the 13th day was distinctly feculent. On the day of admission a careful examination was made by Mr. Cowell and Dr. Hall. The abdomen was examined for tumour, the "rings" for hernia, and the rectum for stricture, all with a negative result. A large enema was administered, with the only effect that a few scybala were washed out. The abdomen was moderately distended, chiefly in the middle line, resonant, and without definite tenderness. The peristalsis of the intestine could be very plainly seen through the thin and flaccid wall, the latter being, as it were, sucked in between the distended coils, and in advance

of the peristaltic wave, causing the convolutions of the gut to be plainly mapped out on the surface. The pulse was fairly good, and the temperature slightly below normal. The vomiting, although of a stercoraceous character, was not urgent.

On the third day after admission, and the fifteenth of obstruction, it was decided to operate, and laparotomy was performed by Mr. Cowell. After emptying the bladder and carbolising the abdominal surface, an incision from 3½ to 4 inches long was made in the middle line below the umbilicus, and the peritoneal cavity soon opened. The presenting lesser bowel was distended and claret-coloured. On introducing the hand into the abdomen the cæcum was found to be empty and collapsed, and several coils of collapsed small intestine were found in the pelvis. The internal rings being next examined, it was soon found that the small intestine was firmly adherent to the orifice of the right crural canal. Here was evidently the occlusion. So intimate was the adhesion, the result of a previous chronic constricting peritonitis, that the gut was acutely bent at this spot, and suggested a partial enterocele into the crural canal. The latter was now cut down upon from Scarpa's triangle; but it contained only an occluded old hernial sac, in the cavity of which were a few drops of a clear fluid. This was pushed with the finger into the abdomen, the intestine freed by division of the sac, after ligaturing in two places, and the remains of the latter returned to the crural canal. The peritoneal cavity having been cleaned out with carbolised sponges, the wound was closed with deep and superficial silk sutures. The patient rallied well after the operation, and there was no vomiting. A little iced brandy and water was at first given, and a small quantity of morphia administered at night hypodermically. During the next day the pulse became very feeble, and the temperature, which in the morning was 98·87°, rose in the evening to 101°. On the morning of the third day delirium was well marked, and the patient's condition became gradually worse, the temperature rising to 105°, and the pulse becoming more and more imperceptible, until death supervened 48 hours after the operation, the immediate cause of the fatal result being pneumonia.

At the post-mortem examination, 25 hours after death, the intestines were found to be distended and of a dark red colour.

The peritoneal surface was, however, fairly smooth and shiny, except in the immediate vicinity of a knuckle of gut just 7 feet above the ileo-cæcal valve. Here there was evidence of incomplete constriction of the intestine, and the gut showed a furrow or depression. Its peritoneal surface was coated with a thin layer of lymph, and the whole thickness of the intestinal wall hereabouts was much increased, and the colour a dark brownish red. The peritoneal cavity contained several ounces of a bloody fluid, but no clots. Examination of the femoral canal showed that a piece of peritoneum (hernial sac) had been cut off, and the ligatured stump returned to the canal. The base of one lung was pneumonic.

Remarks.—This case is a good example of the difficulties which beset the surgeon in attempting to arrive at a diagnosis in cases of obstruction. Although the woman must have at one time suffered from a femoral hernia and local peritonitis, no statement was volunteered on this point, nor by leading questions could any such history be obtained. And again, although there was evidence, in the comparative ease with which a prolonged period of acute obstruction was withstood, in the late occurrence of stercoraceous vomiting, in the fact that the enlarged intestinal coils could be plainly traced on the abdominal surface, and post-mortem in the stenosis of the gut and hypertrophied intestinal walls, that considerable obstruction had existed for some time, no inconvenience apparently resulted, and no symptom occurred to excite attention. The complete occlusion was probably due to an alteration in the position of the intestine causing a more angular bend than had previously existed.

CASE 2. *Strangulation under an adherent appendix.*—Dennis M—, aged 16, was admitted into St. Matthew Ward on September 22nd, 1891, in a collapsed and almost hopeless condition with stercoraceous vomiting, and a temperature of 97·2°. The abdomen was greatly distended, tympanitic, and tender on pressure. Pain was complained of in the right side, extending down to the iliac fossa. The attack began suddenly 4 days before with severe pain in the umbilical and cæcal regions. No cause could be given. There was early collapse, with vomiting, which towards the end became feculent. Constipation was absolute from the first. This lad had been in the hospital on

three different occasions within the last 4 years with symptoms of perityphlitis, attended with constipation. He made an average stay of 12 days, and was discharged recovered each time. The present attack, in symptoms and course, had but little in common with the previous ones, and there was some uncertainty as to the cause of the obstruction. The patient being young, although apparently in extremis, laparotomy was immediately performed by Mr. Cowell, the incision selected being the median one below the umbilicus. The small intestine was found to be greatly distended and much congested; but there was no peritonitis. The hand being now introduced into the peritoneal cavity, the cæcum was found to be empty and the ileum collapsed. On tracing the latter upwards, the source of trouble was soon discovered to be the strangulation of a knuckle of the small intestine, which had slipped under an arch formed by a distorted appendix and a strong short fibrous band connecting this with the mesentery at the right of the lumbar spine. On division of the band the obstruction was immediately relieved, and the fluid contents of the bowel above readily entered the empty ileum and cæcum below. The abdominal cavity having been cleaned out with carbolised sponges, the wound in the parietes was closed with deep and superficial silk sutures. The patient did not rally, and sank two hours after the operation.

Post-mortem 17 hours after death. On opening the abdomen coils of intestine of a dark red colour, mottled with diffuse areas of black, were seen. These coils were found to belong entirely to the upper part of the ileum and to the jejunum. Near the upper end of the ileum the gut was adherent by old adhesions to the vermiform appendix, which in its turn was fixed to the pelvic brim, near the right sacro-iliac synchondrosis. The vermiform appendix made a V-shaped bend on itself, its proximal half being quite occluded by a fibrous growth involving its mucosa and submucosa. Beyond this growth, which was about the size of an almond, the end of the appendix formed a *cul-de-sac* containing a little thin greenish fluid. The mucosa of the lower part of the ileum and of the colon was quite normal; but in the upper half of the intestine, which like the stomach contained thin cocoa-like fluid fæces, the mucosa was swollen, red, and mottled with numerous areas of hæmorrhagic

extravasation. The superjacent peritoneum was smooth and shining. Mesenteric glands large and soft. In the mesentery, and lying between the vermiform appendix and the adherent gut, was a circular aperture about the size of a florin. It had apparently been made by surgical interference (division of band).

Remarks.—Uncertainty of diagnosis existed in this case, owing to the fact that the symptoms of the severer forms of perityphlitis, *e.g.*, those due to perforation of the appendix, closely resemble those due to internal strangulation. The history did not help one much, as the attack might either have been a relapse, in an aggravated form, of the disease that he had previously suffered from, or an internal strangulation made possible by the previous local peritonitis. The abdomen has often been opened for supposed strangulation when the symptoms were due to a perforation of the appendix. It is well known also that the majority of cases of internal strangulation in the male depend on a previous perityphlitis for their cause. The points chiefly relied on in this case in arriving at the diagnosis of acute internal constriction of the gut were the following: vomiting was urgent, and was early of a feculent character, absence of local signs of peritonitis, no special tenderness or fulness in right iliac fossa, subnormal temperature, and early collapse.

CASE 3. *A rare case of volvulus of the colon, with a rapidly fatal result.*—John J—, aged 10, was admitted under Dr. Sturges on March 23rd, 1890, in a moribund condition. He was cold, blue, and pulseless, with an enormously distended and tympanitic abdomen. Subcutaneous injections of brandy and warmth had no effect in the attempt to rally him, and he died in ten minutes. The history of the illness, which was afterwards taken with care, was as follows: The lad was in his usual good health on the previous day, Saturday the 22nd, and after eating heartily at tea-time, went out with a baked potatoe man until 12 o'clock, when he went home. On his arrival he complained to his mother of slight "belly-ache"; but no particular notice was taken of this, and the boy went to bed, sleeping for three or four hours. Between 4 and 5 o'clock in the morning, however, the abdominal pain had so increased in

severity that he called his mother; but even then he soon dropped off to sleep again. At daybreak the pain was much worse, accompanied by great thirst and complete loss of appetite. Turpentine and hot flannels were applied to the abdomen, and peppermint given internally, also a dose of rhubarb and magnesia. Between 12 and 1 o'clock the pain caused the boy to double himself up for its relief, and vomiting began. Everything now taken to drink was immediately ejected, and the vomited matter became bile-stained. He had a great dread of being taken to the hospital, and struggled much when the friends were removing him. He was said by the mother to have passed a small motion in the morning. There was no history of constipation, nor of his having had anything unusual to eat before the attack, and the lad had not previously suffered from colic. The only cause for the illness that the friends could suggest was that in the afternoon of the previous day (22nd), he was seen to fall on his abdomen across the shaft of a cart. He, however, made no complaint, and did not appear to be hurt. Death took place in 15½ hours after the first symptom of the disease.

Post-mortem examination 24 hours after death. On opening the peritoneal cavity, the cæcum, ascending and transverse colon were found to be enormously distended, and of a green colour. The rest of the intestine, both above and below, was normal in appearance and structure. The caput coli was found in the left iliac region. The ascending and transverse parts of the colon, up to within three inches of the splenic flexure, had made a double twist around its mesenteric axis. The meso-colon was unduly long. There was no obvious cause for the volvulus. On opening the affected gut it was found of a dark chocolate colour, presenting several areas where the structure was lost, so that these patches appeared like pieces of thin parchment. The contents of the twisted portion were a brick-red grumous fluid.

Remarks.—Not only does volvulus occur almost exclusively in the sigmoid flexure, but even there it is extremely rare before the age of 20. Treves, in his manual on intestinal obstruction, refers to one case mentioned by Leichtenstern in a child aged 10. Although favoured by a long meso-colon no cause could be discovered for the twist. The fatal result, which in this case was most exceptionally rapid, was probably due in a great measure to the pressure which the immensely distended bowel

exerted on the thoracic viscera. Perhaps the soda water and ginger beer which the mother gave the boy to quench thirst contributed in no small degree to the rapid distension of the gut. One can scarcely imagine more undesirable beverages under the circumstances. Had the boy not died practically before treatment could be thought of, puncture of the gut would probably have afforded some relief.

CASE 4. *Strangulation of a knuckle of the jejunum under an omental band.*—Jessie B—, aged 14, was admitted into Harpur Ward under Dr. Allchin on August 5th, 1890, suffering from acute intestinal obstruction. With the exception of an occasional "bilious attack" and constipation, often lasting a week, the girl was said to have always enjoyed good general health. She had never had any serious illness, nor was she known ever to have suffered from any symptom of abdominal disease before the present attack. Menstruation had not commenced. There was no family history of tuberculosis. Her fellow-servant stated that on July 29th she (the patient) had partaken of some indigestible food, after which she felt unwell. On the 30th she complained of pain in the left iliac region, but she was not particularly ill. On the 31st vomiting began, which was soon bilious in character. On August 2nd, constipation having persisted, a dose of antibilious pills was given, and afterwards Epsom salts, but no relief to the bowels followed, and the vomiting continued, the ejected matter becoming very offensive. There was now great pain in the region of the umbilicus, and the patient passed a sleepless night. On the morning of the 3rd patient stated that a small hard motion, accompanied by much straining, was passed; but the friends denied that there was any action of the bowels. In the evening an enema was administered, which returned mixed with a little blood. This was repeated on the 4th with a similar result. The offensive vomiting persisted, and on the following day the girl was sent to the hospital.

On Aug. 5th, the day of admission and 8th day of obstruction, the symptoms, physical signs, and effects of treatment were carefully observed and noted by Dr. Allchin, of which the following is a short account:—

The girl did not appear to be in much distress. Complained

of nausea, but not of pain in the abdomen. Tongue furred and rather dry. Pulse small and somewhat quick. Heart sounds normal. Temperature 98·6°. Abdomen slightly distended with general tenderness, more marked on the left side. Nothing abnormal to be felt on palpation. A large enema was given, which returned without effect. This was repeated in the afternoon with the same result. Later, great pain was experienced in the left side of the abdomen, and vomiting of distinctly stercoraceous matter commenced. This continued off and on for some hours, and at midnight the pain had increased in severity, and the whole of the left side of the abdomen was markedly dull as compared with the right. Temperature normal. Rectal examination showed, easily within reach of the finger, a condition apparently like that of an invaginated gut; but there was no fæces, mucus, nor offensive discharge brought away on the finger. A grain of opium was given, after which the patient slept fairly well.

August 6th.—Opium pill, gr. 1, repeated at 6 a.m. At 9.30, with the girl on her side and hips well raised, three pints of warm water and oil were slowly injected into the bowel and retained there for a few minutes. This returned unaltered, but was accompanied by flatus. Digital examination of the rectum now showed that the supposed invaginated gut had receded, and was only just within reach of the finger. Opium pill, gr. 1, again at midday, and an hour later injection of three pints of warm water and oil repeated with the same result as before, and flatus was again passed. Ice was given to suck, and nourishment consisted of beef-tea made from Liebig's extract, and given in teaspoonful doses. In the evening the enema was repeated without effect. Opium pill, gr. $\frac{1}{2}$, ordered to be given every eight hours.

August 7th.—The stercoraceous vomiting continuing, with persistence of absolute constipation, Mr. Spencer was asked to see the girl, with the view of having an exploratory laparotomy performed if necessary. He noticed the fulness in the left iliac fossa, and also per rectum, the fold of gut having the feel of an invagination. The girl having been removed to the theatre, and chloroform administered, a further examination was made, and before proceeding to operative measures it was thought advisable to repeat the injections while under an anæsthetic. This was

accordingly done with warm water and oil, in rather smaller quantities than before, and after four or five repetitions several largish lumps of light-coloured fæces with flatus were passed. With this result it was decided to postpone operation, and the girl was taken back to bed.

August 8th.—Condition worse. Fæcal vomiting more persistent. No action of the bowels. Pulse 120, regular, soft, and quick. Temperature varying between 97° and 97·6°. Distinct fulness in left iliac fossa. The urine was not measured, but it was small in quantity and contained albumen.

Operation.—The skin of the abdomen having been rendered aseptic by the application of carbolised lint, and finally by sponging with carbolic lotion, 1 in 20, and the bladder emptied, an incision about 4 inches long, having its centre opposite the anterior superior iliac spine, was made in the left linea semilunaris, and the peritoneal cavity opened. The intestines which bulged up into the opening were congested, but there was no peritonitis. The hand was now introduced and the left iliac fossa, colon, cæcum, and internal rings examined, but nothing abnormal was found. On tracing upwards the small intestine, after slightly enlarging the incision in the parietes, the gut was felt to be fixed at the right of the lumbar spine, low down. Some coils of intestine which escaped from the wound were immediately enveloped in warm carbolised cloths, and the fixed portion more closely examined. It was soon seen that the occlusion was caused by the strangulation of a knuckle of the jejunum under a tense, short band, the result of a previous peritonitis. So severe had the constriction been, that the gut had recently ulcerated through, with extravasation of some fæces, and the setting up of local peritonitis. After ligaturing the band in two places, it was divided in the middle, the two ends springing back like a piece of tense indiarubber. The gut, which was found to be gangrenous, was grasped and drawn well outside the abdomen, and the other coils replaced after very carefully sponging out the peritoneal sac. The disease being high up in the jejunum, Mr. Spencer decided to resect the gut at once. The intestine having been clamped well on either side of the affected portion, a piece of the bowel about 3 inches long, together with a triangular piece of the mesentery, was removed. The upper cut end of the gut was now pushed into the lower, the edge of the

latter being first turned in, the part now having the appearance of an intussusception. Before this could be done, however, it was necessary to stiffen and reduce the calibre of the dilated upper end, by introducing into its lumen, and stitching to its mucous membrane, an indiarubber tube about an inch long and of proper diameter, made from a piece of ordinary indiarubber tubing by slitting and rolling longitudinally, and securing in apposition the two ends by a stitch or two. The peritoneal surfaces of the divided ends were united together by a number of fine catgut sutures, the tube being of course left inside to be passed at a future time if the patient lived. The gut was now well cleansed and returned to the abdominal cavity, the wound united by deep and superficial silk sutures, antiseptic dressing applied, and the girl removed to bed. She, however, did not rally, and survived the operation only about three hours.

Post-mortem examination 21 hours after death. Body emaciated. Post-mortem decomposition marked. On opening the abdomen 2 or 3 ounces of blood-stained fluid were found in the pelvic cavity. The peritoneal surface was everywhere either of a dark brownish red or of a greenish colour. The upper part of the gut was somewhat distended and the lower part contracted, but contained some soft clay-coloured fæces. About 45 inches from the pylorus the gut had been completely divided, about 3 inches having been removed, and the upper and lower ends united quite firmly by catgut sutures. The constricting band was found to have been a piece of the great omentum, which had almost surrounded the constricted jejunum, binding it down close above and to the inner side of the caput coli, close to the ileo-cæcal valve, the remains of the constricting band, as shown by the catgut ligature, being found in this place. Just $1\frac{1}{2}$ inch from the valve the ileum had evidently suffered from pressure, as at this part its calibre was much diminished. The peritoneum was everywhere slightly blurred from a thin layer of recent lymph. It also showed that it had on some previous occasion been acutely inflamed, as there were not only numerous adhesions (in addition to the omental band) about the spleen and in the pelvis, but also several encapsuled collections of caseo-cretaceous matter. The uterus was almost rudimentary, and was firmly adherent to the bladder. The ovaries were also bound down by chronic peritonitis. In the vicinity of the uterus were

two of the caseo-cretaceous collections alluded to. The stomach was normal.

Remarks.—This is yet another example of the difficulty which is generally experienced in attempting to arrive at a differential diagnosis in this class of cases. The symptoms did not at all agree with obstruction of the upper part of the jejunum, excepting perhaps the absence of marked meteorism. Moreover, there was the evident fulness and dulness of the left iliac region which eventually led Mr. Spencer to open the abdomen in the left semilunar line. The girl had evidently, at some previous period, suffered from acute or subacute peritonitis; but the symptoms had not apparently been sufficiently marked to attract the attention of the friends. The caseous nodules were not connected with the mesenteric glands, and there was no evidence of previous gland disease.

A CASE OF EXTENSIVE HYDATID DISEASE OF THE PERITONEUM.

REPORTED BY MR. POWELL.

MARY C—, æt. 25, single, was admitted into Hollond Ward, on October 19th, 1891, suffering from a large abdominal tumour. It appeared that she had had a child six years before, from which time she dated the commencement of her trouble. The abdomen, according to the statement of the mother, did not properly assume its natural size after the confinement, and the patient was never afterwards entirely free from some abdominal pain or discomfort. About a year after childbirth the patient noticed that her abdomen was distinctly distended, and that she periodically—about once a fortnight—suffered much pain, with a feeling of pressure, and noticed some difficulty in walking. Menstruation was regular, but gradually diminished in quantity. During the last two years the tumour steadily and more rapidly increased in size, with periodical pains of a more severe character, confining the patient to bed for from a few days to a week at a time. Constipation was towards the end a prominent symptom, and enemas had often to be resorted to for the relief of the bowels. Some difficulty of micturition was also complained of.

On admission the patient was carefully examined by Mr. Cowell and Dr. Potter. The abdomen was irregularly distended, being very prominent in the umbilical region, dull on percussion, bossy, and fluctuating, conveying the impression that the tumour was made up of a number of separate cysts. Per vaginam several similar fluctuating tumours could be felt in the pelvis, and the uterus was more or less fixed and raised unusually high up. One of the cysts at the most prominent part of the swelling near the umbilicus was aspirated and a clear fluid withdrawn, which, however, was not examined microscopically.

Operation being decided upon, the patient being very anxious that something should be done, laparotomy was performed by Mr. Cowell, the incision being the usual one between the umbilicus and the pubes. On opening the peritoneal cavity a large cyst projected into the wound. This was tapped, as also were several others which crowded into the opening. Some very dense adhesions required division before the hand could be passed upwards along the parietal peritoneum. The abdomen was now found to be literally filled with cysts of all sizes, from a marble to a goose's egg or larger. In shape the smaller ones were spherical and the larger ones oval, with one end directed towards the anterior abdominal wall. The latter were exceedingly tense, and some of them were ruptured in the attempt to remove them. The outer membrane was as thin as parchment, smooth, semi-transparent, and very tough. Within this adventitia was the true cyst wall—almost white, elastic, about as thick as stout chamois leather, easily torn, and readily collapsing when ruptured. The contained fluid was watery, of specific gravity 1010, neutral, no albumen, and with the usual excess of chlorides. The deposit placed under the microscope was found to consist of innumerable tænia-heads and hooklets. The very young cysts were lying almost loose in the altered peritoneal cavity. They were limp, easily ruptured, and appeared to be devoid of adventitia. This, however, was probably only apparent, for on examining cysts of different stages of development, from the immature to the mature, the adventitious capsule became more and more recognisable. To return to the operation: from twenty to thirty, counting only the larger ones, of these cysts were removed. The majority could be easily shelled or torn out of the loose areolar bed in which their bases lay, especially those situated in the omentum. Many of them were removed by placing a number of ligatures and cutting off the greater part of the omentum. The pelvic cavity was found to be crammed with these growths, and it was not found practicable to remove them. These, with others deeply seated in the posterior part of the abdomen and under the diaphragm, were therefore left, and after washing out the peritoneal sac with warm water, the wound was closed with deep and superficial silk sutures, a large glass drainage tube being placed in its lower angle. The patient never really rallied from the shock of the operation, and died in 30 hours.

Post-mortem examination.—Some bloody purulent fluid was found in the abdominal cavity. Intestinal coils matted together by old adhesions. The peritoneum was everywhere, but chiefly in the pelvis, the seat of a large number of hydatid cysts, from the size of a pea to that of an orange. The liver contained three cysts of considerable size, one in the right lobe, one in the left lobe, and one with a shrivelled, collapsed capsule about the middle of the inferior surface. The right kidney was flattened from neighbouring cyst. The uterus was fixed from old inflammation and presence of cysts.

Remarks.—It will be seen by the post-mortem notes that the liver contained the remains of a ruptured cyst. The dissemination of its contents was, then, the probable cause of the general peritoneal disease. Rupture and peritonitis possibly occurred during and after labour, for the patient was said to have had a very bad time, and was confined to bed for a considerable period. The drinking water was obtained from a pool in the village, surrounded by a simple rail. No other case of hydatid disease has been known to have occurred in the neighbourhood. The evident existence of adhesions may be held to have contraindicated an operation, and if the hydatid nature of the disease had been recognised, the cysts could have been attacked *seriatim* with some temporary success. But the miserable condition of the patient, her youth and earnest desire for operation, and the possibility of the case being one of multilocular ovarian disease, were factors in determining the attempt to relieve her by operative interference. In any case life could not have been much prolonged.

TWO SURGICAL CASES.

REPORTED BY MR. MILLS.

CASE 1. *Case of Ligature of Right Common Carotid for Aneurism.*

Alfred G—, æt. 43, was admitted into St. Luke Ward under Mr. Macnamara, with a pulsating swelling in the right side of the neck, on July 20th, 1891.

Previous history.—Has been a soldier in active service until three years ago, since then he has worked as a labourer in a coal-yard. His work was very heavy, involving a good deal of lifting and straining. Was much exposed to weather, often getting wet through. Has always been a heavy beer drinker, and when in the army drank freely of spirits. Had syphilis 22 years ago; was under treatment only 2 months. Has had bronchitic attacks during the last few years. Bowels have always been regular. Has not suffered from rheumatism or gout.

History of present illness.—Six weeks before admission the patient was taken ill with vomiting and coughing, coming on at odd times with no obvious exciting cause; this lasted for about 3 weeks. At times it was very severe, so much so that patient had to take to his bed. Three weeks ago he first noticed a "lump" in his neck, about the size of a walnut. Was coughing at the time, and felt something give way. This swelling has gradually got bigger until the time of admission.

Condition on admission.—There is a swelling above the middle third of right clavicle, extending upwards in the neck; above and to the front it extends beyond the anterior portion of the right sterno-mastoid muscle; below it is covered by the sterno-mastoid. Pulsation, expansile in character, is marked. A systolic murmur is heard on auscultation, also there is a distinct systolic thrill. The temporal pulse on the right side is smaller than on the left. The radial pulse on the right side is slightly

smaller than on the left. There is no dysphagia and no congestion of the right side of the head and face. The pupils are equal. The heart's apex is in the normal situation, sounds clear, no murmur. Urine acid, 1024, no albumen, urates, trace of sugar. No other symptoms of diabetes.

On July 25th a needle was passed into the aneurism from before backwards, and the posterior and inner surfaces of the sac were scratched with the needle.

August 1st. The swelling was larger, but it felt firmer. However, it was decided that an operation should not be longer delayed, and the common carotid on the distal side was tied with a silk ligature. After the operation the swelling became distinctly harder.

On August 6th a faint blush was noticed about the wound. This blush extended and the parts became œdematous, and on August 8th a discharge of pus came away. The suppuration of the sac persisted, but the wound nearly closed up until August 17th, when there was only a very small portion not healed. On that day there was profuse arterial hæmorrhage from the wound; this was quickly stopped by firm pressure made by elastic bandage applied over a pad of lint.

August 19th. The wound was dressed at 1.30, when there was no bleeding, but at 3 p.m. profuse hæmorrhage again occurred. The dressing was soaked immediately, and on being removed blood was seen spouting as far as 6 or 8 inches. Digital pressure applied, and the patient was removed to theatre.

Mr. Macnamara being absent from town, Mr. Davy tied the common carotid below the aneurism, there being about ¼ inch of apparently healthy artery below the sac. The wound had to be dressed the same evening, owing to patient having vomited over the dressings. From this date until August 25th, the patient went on very satisfactorily. There was still some suppuration daily from the incision of the first operation, the wound having closed up on the surface, leaving an imperfectly drained abscess cavity. In consequence of this the temperature of the patient rose, and he was delirious for several nights. A tube was introduced, and the cavity was washed out more frequently and the symptoms subsided, the patient making fairly good progress until September 11th, when the temperature again rose and the patient was not nearly so well.

On September 12th the temperature rose to 105°, and the man had a slight rigor. On examination of the wound the opening was found to have again contracted, and it was enlarged and the washing out more effectually carried out. The cavity was much smaller. The first wound for the ligature of the common carotid had not quite healed, there being still a small sinus at the upper and internal angle of wound.

On September 22nd the temperature has continued to go up and down and is somewhat pyæmic in character, and to-day the patient had a slight rigor.

The next day he complained of some pain in the right eye, and there was some swelling of the conjunctiva, with some conjunctival discharge. This condition gradually got worse during the next few days, the eyeball and conjunctiva becoming more and more swollen, proptosis very marked, and the eyelids became œdematous. Mr. Cowell then had charge of the patient, and diagnosed pyæmic ophthalmitis. It was fomented freely, and rapidly suppurated. The eyeball subsequently shrank up, and the swelling and inflammatory condition having subsided, on October 20th the globe was excised. The wound rapidly healed, and the patient speedily improved in his general health, and on November 15th was discharged. He is now attending as an out-patient. There is still a small sinus in the track of the first incision, that for the distal ligature of the common carotid. The wound made at the second operation is quite healed. At the time of his discharge there was no pulsation at the site of the aneurism.

CASE 2. *Case of Hæmatomyelia of the Lower Cervical Region; Dislocation of Fifth Lower Vertebræ (?). Recovery.*

William F—, æt. 13, admitted at 8 a.m. on October 19th into Henry Hoare Ward, under Mr. Macnamara, in a collapsed condition but quite conscious, after falling from a tree. He had been up the tree after a football, and had fallen 40 feet to the ground, striking himself on his back between his shoulders.

There was on admission complete loss of power and sensation in both legs, with deficient action of the intercostal and abdominal muscles; there was no displacement of the spinous processes and no crepitus made out, but great pain on pressure

over lower cervical spinous processes. The face and extremities were blue and cold, pulse extremely feeble. Respiration very shallow, the thorax moving very slightly. He suffered great pain if any attempt was made to move the head or neck.

Within an hour of admission the patient had partly recovered sensation in both legs, and was able to move the left leg a little. Knee-jerks were both exaggerated. There was marked hyperæsthesia over the region between 4th and 6th ribs, the weight of his shirt if moved causing him acute pain. Both pupils were contracted. There was no vomiting nor any mental affection. At 7 p.m. the patient, being kept quiet on his back and lying straight on the bed with the head low, had improved somewhat. Pulse was better, but occasionally irregular. Extremities warmer. There was marked improvement in movement of left leg, but the right was still completely paralysed. The sensation had improved still more, and now seemed normal in both legs and over the abdomen. Breathing was still purely diaphragmatic. The urine continued to be drawn off by catheter. Priapism well marked. At 12 p.m. the patient was still improving. He can move the right leg a little, and the left is better. Priapism continues. Breathing quite shallow and diaphragmatic. Complains of radiating pains from the upper dorsal vertebræ to the front of chest. Zone of hyperæsthesia very well defined as before. Temperature 100·2°.

October 30th, 12 noon. Patient better; moves left leg now quite freely, and right leg is much improved. Priapism continues. Urine drawn off. Radiating pains less marked, also the hyperæsthesia less. At 9 p.m. the patient, lying on his left side, has been sleeping all the afternoon. Hyperæsthesia much less marked. Movement in both legs not so good.

October 31st. Patient better. Has passed urine voluntarily. Moves his legs much better. The priapism has disappeared, and the pain is much less. Sensation over the chest is about normal. The breathing is deeper and the chest moves better.

November 1st. The patient is kicking his legs about, and is much better. He is still kept lying as much as possible on his back. He is very bright and cheerful, and the pain has disappeared. From this date the patient progressed very rapidly, but was still kept in bed lying as quietly as possible.

On November 28th a plaster of Paris jacket was put on, and

the patient was allowed to get up. He soon recovered his strength, and has been sent to a convalescent home.

On January 9th the patient presented himself at the Hospital after being at a convalescent home. He was very well in general health. On examination of the cervical vertebræ a depression was noticed above the 6th spinous process, as if the 5th vertebra had been displaced forwards, or the arch of the vertebra had been fractured.

TWO OBSTETRIC CASES OF INTEREST.

REPORTED BY MR. CATO.

No. 1.—*Case of Papillomatous Cyst of the Ovary.*

Elizabeth B—, æt. 34, married, was admitted July 2nd, 1891, under Dr. Potter, complaining of swelling and pain in the lower part of the abdomen.

Catamenia commenced æt. 17; was very irregular until marriage, which took place at the age of 24. Ten months afterwards she gave birth to a son, the labour being tedious but natural. There was no other pregnancy. Catamenia reappeared nine months after confinement, and continued regular up to admission, coming on every four weeks and lasting three days.

She was quite well until fifteen months before admission; she then noticed a lump about the size of a small egg in the right iliac region. This gradually increased in size, and for some months she considered she was pregnant. Two months and a half before admission she had an attack of "inflammation of the bowels"; suffered from vomiting, diarrhœa, fever, and great abdominal pain. (This was probably the time at which the cyst suppurated.) Since then she had lost flesh, the tumour had increased more rapidly in size, and had extended over to the left side. Had suffered considerably from aching pain across the back and down the legs, and also from a burning and pricking sensation in the vagina. This was aggravated by standing long or walking. She complained of a frequent desire for micturition, and that the act was occasionally accompanied by pain.

Previous history good. Has always been strong and healthy. Family history good.

The last catamenial period before admission commenced June 18th. On admission she was of healthy appearance, but rather thin; tongue clean and moist; respiratory and circulatory systems

normal. Temperature normal. On examination of the abdomen, the tumour was found to be symmetrical, reaching one finger's breadth above the umbilicus. The greatest circumference of the abdomen (31 inches) was through a point midway between umbilicus and symphysis pubis. The tumour was of firm consistence, freely movable, and distinctly fluctuating. There was complete dulness on percussion over the whole tumour, which did not alter on shifting the patient. Good resonance in the flanks. On vaginal examination, the cervix was found to be in normal position; os small; anterior cul-de-sac normal; posterior a little diminished. Uterus not very movable. Impulse through the anterior cul-de-sac was better marked than through the uterus itself. The sound passed in a posterior direction to the depth of 1 inch; would not pass further in any direction. The urine was acid, contained a trace of albumen; specific gravity, 1020.

The patient was examined on September 3rd in consultation by Dr. Potter and Mr. Stonham, and the tumour concluded to be a multilocular ovarian cyst, free from any extensive peritoneal adhesions. Abdominal section was therefore decided upon.

On September 6th, ether having been administered, Mr. Stonham made an incision in the linea alba, between the umbilicus and pubes, about three inches in length. After the peritoneum was opened the tumour immediately presented. It was drawn up into the wound, sponges were packed round it, and a Spencer Wells' trocar thrust in. About 60 oz. of purulent-looking fluid escaped; this was afterwards examined under the microscope and found to contain numerous pus cells and altered blood corpuscles, alkaline in reaction. The cyst wall was then drawn out, and was found to be connected with the right ovary. Two or three broad bands of adhesion were present; these were tied and removed. The pedicle was ligatured and returned after the mass had been cut off. A description of the latter is subjoined. The peritoneal cavity was sponged out until the sponges returned quite clean, and then the edges of the wound were brought together with silk sutures, and the dressings applied. The patient's condition was very good on her return to the ward. She slept a few hours the same night after receiving a hypodermic injection of morphia. The next day, September 7th, she vomited several times (this probably being due to the anæsthetic), and

complained of thirst and a feeling of flatulence; the latter relieved by ol. cajaputi. The diet consisted of beef tea and iced milk alternately at frequent intervals. The temperature rose in the evening to 99·6°, this being the highest point reached after the operation. The urine had to be drawn off by catheter, and still contained a trace of albumen.

On September 8th she felt very comfortable; had no more vomiting. Pulse 76; tongue clean; temperature normal. From this date the patient made an uninterrupted recovery; the urine had to be drawn off until the 10th; she was given fish for dinner on the 14th. The wound was dressed for the first time on the 16th; it had healed entirely, and sutures were removed. On the 20th the dressings were left off; catamenia commenced this day and lasted until the 23rd. Patient got up on the 28th, wearing an abdominal belt with a pad over the scar, and was discharged on September 29th feeling quite well.

Description of Tumour by Mr. Stonham (Specimen No. 1,033 in Westminster Hospital Museum).—This is a papillary cystoma of the ovary. The inner surface of the tumour (outer one as specimen is mounted) is over half its surface thickly beset by sessile and pedunculated papillomatous growths, somewhat resembling in general appearance the head of a cauliflower. All stages in development of these can be seen, from those of the size of a pin's head upwards. On the outer surface of the tumour the enlarged ovary and the divided pedicle are present, with two or three broad bands of adhesion. The cyst wall is thickened, and the outer surface is smooth, shiny, and of a dead-white opaque colour. Microscopical examination of the papillary masses shows them to be composed of fibrous tissue covered with cubical epithelium. Many of the processes are undergoing cystic degeneration, so that a section is studded with small cysts; some of the epithelial cells are colloid.

No. 2.—*Case of Malignant Disease of the Omentum and Peritoneum.*

Caroline S—, æt. 55, was admitted under Dr. Potter on September 29th, 1891, suffering from pain, swelling of the abdomen, and tenesmus. Had been in fairly good health until six weeks before; she then noticed a small lump in the abdomen to the right of the umbilicus. Fourteen days after this she

began to suffer from constipation, tenesmus, and diminished micturition, which symptoms continued up to admission.

Fourteen days before admission general swelling of the abdomen began to manifest itself, and, as this increased, the tumour in the right iliac region became less distinct. She stated that she had "inflammation of the bowels" when a child. Had been suffering for past seven years from winter-cough. Had an attack of dropsy of the legs and abdomen in 1889, which only lasted a few weeks.

On admission she was emaciated, suffering considerably from dyspnœa. Expression anxious; respirations 30; pulse 90; temperature 99°. Chest hyper-resonant; heart dulness partly overlapped; no added sounds. Abdomen moderately distended, measuring 34 inches in the greatest circumference; complete dulness over the lower half of the hypogastrium, no dulness in the flanks. There was increased resistance on the right side of the umbilicus, and on sharply pressing the hand down in this region it came on a hard mass which immediately receded. Obscure fluctuation was felt in the hypogastrium. A catheter was passed, drawing off 2½ ozs. of urine, which contained a large amount of urates and no albumen. This made no alteration in the dulness. On vaginal examination the cervix and os were found to be small, pointing posteriorly; the uterus was light and movable. There was a band of fibrous tissue extending across the posterior fornix. The patient was ordered digitalis, citrate of potash, and infusion of scoparia, but until October 10th the daily amount of urine only averaged 9 ozs.; being all the time free from albumen. During this period the distension of the abdomen had been increasing very rapidly; on October 5th the measurement being 36½ in.; on October 8th, 37½ in.; and on October 12th, 38½ in. On October 7th the cough became more troublesome, and she vomited several times. The abdomen was very tense and painful. Some moist sounds could be heard at the extreme bases; no dulness. The next day there was slight general bronchitis, the moist sounds being heard over the whole chest. General condition much worse; marked orthopnœa and some cyanosis of the face. The tumour could still be felt in the right iliac region, and appeared to be larger.

On October 10th Mr. Macnamara examined the patient with Dr. Potter, and an operation was decided upon.

On October 12th chloroform was administered, and Mr. Macnamara made an exploratory incision, three inches in length, below the umbilicus. On opening the peritoneal cavity some dark blood-stained fluid escaped; in all 6 pints 16 ounces. On introducing the hand into the abdomen a large diffused nodular tumour could be felt, movable below but fixed above, extending for some distance on either side of the middle line but chiefly situated to the right. Secondary growths were found, infiltrating the parietal and visceral layers of the peritoneum, the primary growth apparently involving the omentum. When the edges of the abdominal wound were held apart the growth could be clearly seen, and had all the appearance of colloid carcinoma. The abdominal cavity was thoroughly irrigated with warm water, a glass drainage tube inserted, and the abdominal incision sewn up.

The operation lasted half an hour, the pulse and general condition of the patient at the end being very good. She made a good recovery from the shock of the operation, and was considerably relieved by it, being able to lie flat even on the following day. She suffered for several days from slight bronchitis and occasional sickness, but gradually regained her strength and got up about three weeks after the operation. The daily amount of urine had increased two days before the operation to 23 ozs.; this was maintained afterwards, on some days being as much as 30 ozs., and always free from albumen. When the abdominal wound had healed sufficiently to allow of an examination, the growth was found to be rapidly increasing, extending laterally into both flanks and upwards towards the epigastrium. There was no re-accumulation of fluid, but the abdomen was becoming gradually distended with this solid growth. The bowels had been kept open every three days with simple enemata until a few days before her discharge, when an aperient pill every night was found to suffice.

The patient was discharged on November 23rd, the abdomen being about the same size as on admission, but causing her very little inconvenience and absolutely no pain. The growth felt hard and nodular, extending over the whole of the lower part of the abdomen and upwards to a point midway between the ensiform cartilage and the umbilicus. By vaginal examination the uterus was found to be firmly fixed, and the cul-de-sac hard and shallower than before.

AN EARLY CASE OF LEPROSY.

UNDER THE CARE OF DR. P. S. ABRAHAM.

REPORTED BY MR. CAMPBELL.

A West Indian boy, aged 7, was admitted into the West Extra Ward on May 13th, 1891, under the care of Dr. Abraham, suffering from the macular form of leprosy.

The history was this: The boy was born and had lived all his life in British Guiana, both his parents being natives of that place; he had always been strong and healthy, and, with the exception of measles, had never had any illness.

His friends state that they can trace the family back for some generations, and that they cannot find any member who had suffered from any skin complaint.

The present illness is said to have begun in the earlier part of the year with a feverish attack, which was not marked by any serious symptoms, but in a few days a red spot was noticed just internal to the left nipple, and this was followed by a uniform swelling and redness of the right ear, which seemed to be growing larger than the left, and also by the appearance of a reddish spot in the middle of the left cheek. These three spots came out in the course of a week, and in the ensuing fortnight many other similar spots were noticed on the arms and hands, especially the left, and on the buttocks and legs. The boy was seen by two local medical men, and the diagnosis was that he was the subject of leprosy. He was in consequence immediately sent to England.

As far as can be learnt from a friend of the patient's, who is in England, the boy was vaccinated for the first time about a year previous to the present illness, calf-lymph being used. At the present time he has two vaccine cicatrices on the upper part

of the right arm; the cicatrices are about the size of a shilling, white in colour, with a ring of pigmentation round the edges.

On admission the boy, who has a dark skin, was covered with many slightly raised, thickened spots, varying in size, and some of which seemed to be desquamating. On the left cheek there was a circular spot about one inch in diameter, of a bluish-red colour, slightly raised and indurated, and shiny looking; around this spot, and over the bridge of the nose and just below the right eye, there were similar spots, only much smaller in size. The whole of the right external ear was larger than the left, and of a dull red colour, the swollen integument not being markedly indurated. Over the left breast there was a circular patch about the size of a five-shilling piece, the centre of which was quite white and the ring around of a dull red colour; it was not raised, but had a rather shiny appearance. On the left arm there were two large patches, one on the front in the bend of the elbow-joint, and the other on the back of the arm just above the elbow; both these patches had similar characteristics to the other large patches. On the left hand the skin over the thumb, thenar eminence, and index finger was of a dull red colour, shiny, and slightly raised; there was also some thickening about the proximal phalanx of the thumb and first finger, the patient being unable to entirely flex them; it was also noticed that the patient did not make use of them. There were several smaller spots on the right arm varying in size from a mere speck to a sixpenny piece; they were very slightly raised, of a brownish-red colour, and were not indurated. There were several spots over the buttocks, front and back of the thighs and legs, and also on the dorsum of the feet, but none on the plantar aspect. Just above the right knee there was a large-sized patch, having the same characteristics as the other patches. The smaller spots are about the size of a sixpence.

There was no subcutaneous induration to be felt over the course of the ulnar or external popliteal nerves, nor any obvious wasting of the muscles supplied by them, nor did the patient complain of any pain. No definite anæsthesia could be made out, his answers being contradictory. The general impression was, however, that there was some change of sensation. There were no spots in the mouth. A physical examination of the lungs and abdomen indicated nothing abnormal.

On May 14th patient was injected with ½ milligramme of

Koch's tuberclin. There was no marked reaction, but the patient seemed rather drowsy after the injection, and the temperature gradually fell about 2°.

On May 16th he was injected again with 1 milligramme, but there was no reaction, except that the temperature fell about 1·5°. It was thought, however, that the spots became rather more apparent.

On May 18th he was injected with 3 milligrammes; this was followed in 15 hours by a rise in temperature, which reached 101° in about 24 hours, the pulse and respirations being very frequent. Patient complained of pain all over him, seemed much depressed, took his food badly, and there was occasional vomiting. Many of the spots on the skin became more marked, and there was much sweating. On the 19th the temperature had reached 103·2°, the pulse being 148, and the respirations 36. The temperature then gradually fell, reaching normal on the 21st, but the patient seemed very weak and prostrate.

On May 26th he was injected with 2 milligrammes, but there was no definite reaction; the patient seemed drowsy, and the temperature gradually fell 3°.

On May 29th he was injected for the fifth time with 3 milligrammes; the temperature fell 1°, and he seemed drowsy. Several of the spots, especially those on the left cheek and left breast, seemed of a deeper colour and more prominent.

On June 4th he was again injected with 5 milligrammes, the temperature falling about 1°, but there was no reaction, and the spots remained about the same.

In the course of the ensuing 10 days, however, all the spots became much more prominent, of a darker colour, and more indurated.

The right ear became much more red and swollen, the redness extending on to the skin both behind and in front of the ear.

The left hand also became worse, the swelling and redness extending further over the dorsum, the thumb and index finger were more swollen, and it had spread to the middle of the middle finger, the patient being unable to flex the fingers. He, at this time, did not take his food so well, and had a return of incontinence of urine, which was first noticed on admission. The patient was taking oleum gynacardia, minims i, in capsules, three times a day.

On June 19th he was injected with 5 milligrammes; he was very drowsy, but there was no reaction, the spots remaining much in the same swollen condition. A few small fresh spots, however, were noticed.

On June 22nd many of the spots seemed decidedly less prominent, and the left hand and right ear were less swollen. The dose of oleum gynacardia was increased to 2 minims three times a day.

On July 8th he was injected with 6 milligrammes; he seemed very drowsy, and slept soundly for 1½ hours. The temperature fell about 1° some 5 hours after the injection. There was no difference in the appearance of the spots.

On July 9th Dr. Abraham made an incision into the indurated tissue about the proximal phalanx of the left index finger, the skin having been previously painted with a 10 per cent. solution of cocain; a small piece of tissue and some serum were removed; the patient did not appear to feel any pain. On microscopical examination no bacilli could be found.

On July 11th a small indurated spot was excised from the back of the right thigh for microscopical examination. The skin was first painted with a 10 per cent. solution of cocain, but the patient seemed to feel a great deal of pain.

On July 18th and 21st he was again injected with 8 and 10 milligrammes respectively, but there was no noticeable reaction; he was very drowsy; the spots seemed to be much less prominent.

After the injection on July 21st he was not injected any more, but was kept on the oleum gynacardia, minims 3, three times a day.

On making an examination of the patient in September, it was noticed that very many of the smaller spots had entirely disappeared, and the larger patches were less swollen and indurated. The right ear seemed quite normal, and the red ring on the left breast was much less, the white centre having increased; no anæsthesia, however, could be made out over this white area. The swelling of the left hand had quite disappeared, but there was still some discoloration of the skin, over which there seemed to be slight anæsthesia. He was able to completely flex his fingers.

On several occasions the patient's blood was examined microscopically, both by Dr. Abraham, and also by Dr. Hebb, the

Pathologist to the Westminster Hospital, but bacilli could never be found.

Remarks by Dr. Abraham.

This case is of interest from several points of view. If the history be correct, the boy was in perfect health, and without a spot upon his body, five or six weeks before he came under my notice. The diagnosis was confirmed, before the patient left Demerara, by Dr. Castor, the well-known Medical Superintendent of the Mahaica Leper Asylum; and shortly after the boy arrived in this country, I believe that he was seen by Dr. Radcliffe Crocker and Professor Curnow, both of whom appear to have had no doubt as to the nature of the case. The friends in London were in the greatest difficulty as to what should be done with him, and were recommended to apply to me by Dr. Curnow. Through the kindness of Dr. Donkin, the poor child has had the advantage, for several months, of treatment and shelter at the Westminster Hospital, pending the arrangement for him of a permanent domicile.

It is not often that such early cases of leprosy come under our notice; and seeing how accustomed we are to look upon a "leper" only as a repulsive or mutilated object, it is not to be wondered at that, at first sight, doubt may arise as to this case being one of leprosy at all. Even in countries where the disease is endemic, and the local medical men have frequent opportunities of seeing cases, the early stages are often overlooked, the symptoms being so trivial that the patients at first do not come under medical care, or being perhaps put down to syphilis or other complaints.

The history of this case, in many respects, is in accordance with what we know of leprosy. The patient was born, and lived, in a country where the disease is not only endemic, but probably spreading; his affection commenced with a prodromal fever,* which was soon followed by an eruption of erythematous patches, some of them of a large size, and remaining for many months, on the face, limbs, and other parts of the body. Some of these spots showed subsequent dusky pigmentation, and one of them became leucodermic in the centre. The enlargement of the right

* Some authorities do not admit that a pyrexial attack is a necessary prodroma of leprosy, and I have myself come across cases in which there was absolutely no history of anything of the kind.

ear, especially of the lobe, and the obvious loss of power of the thumb and forefinger of the left hand, were also characteristic symptoms. Since the swelling of the latter has gone down, a wasting of the thumb and forefinger muscles has become apparent. As mentioned by Mr. Campbell, we could never get very satisfactory evidence as to anæsthesia, either of the spots or of the affected hand, the child's answers always being more or less ambiguous. On one occasion when, with his eyes averted, he was touched on the finger with the bottom of a test-tube filled with hot water, he said it was "cold," but on repeating the experiment in a few seconds with the same tube, he said he thought it was "hot." When an incision was made in the skin of the finger, which, however, had been previously painted with a 10 per cent. solution of cocain, he certainly showed no signs of feeling it in the least—a very different result to what happened when the macule on the thigh was excised. Although the skin was similarly painted with cocain, there was here abundant evidence of no anæsthesia.

The fact that the characteristic bacilli of leprosy were not found either in this piece, in the skin of the finger, or in blood obtained from a prick on several occasions, is, possibly, of some negative value. But we must remember that, although we never fail to find bacilli in the leprous nodules, nerves, and often in other tissues, in well-developed cases, examinations for bacilli have not as yet been frequently made in such very early cases—in which some of the lesions, at any rate, may be regarded as nutritive rather than neoplastic.

I desire to record my thanks not only to Dr. Donkin for so kindly giving up the use of one of his beds, but to Drs. Gossage and Sharman, and particularly to Mr. Campbell, for the skilful care and attention which they devoted to the case.

OFFICIAL REPORTS.

REPORT

OF THE

POST-MORTEM EXAMINATIONS MADE IN 1890.

By Dr. HEBB.

Cases recorded		231
Males	142	
Females	89	
General Diseases:		
Erysipelas	1	
Diphtheria	5	
Septicæmia and Pyæmia	6	
Delirium tremens	1	
Rickets	2	
Tuberculosis	21	
	—	36
Diseases of the Nervous System:		
Cerebro-spinal meningitis	7	
Hydrocephalus	2	
Abscess of brain	1	
Hæmorrhage into brain	3	
Softening of brain	2	
Tumour of brain	1	
Convulsions	4	
	—	20
Diseases of the Ear:		
Otitis	2	
	—	2
Diseases of the Circulatory System:		
Endocarditis	16	
Aortitis, including aneurysm of aorta	6	
Pericarditis	4	
Congenital heart	1	
	—	27
Diseases of the Respiratory System:		
Bronchitis, acute	1	
Broncho-pneumonia	13	
Pneumonia	14	
Gangrene of lung	1	

Diseases of the Respiratory System—*continued.*		
Bronchitis, chronic, and emphysema	8	
Phthisis	15	
Empyema	5	
	—	55
Diseases of the Digestive System:		
Epithelioma of lip	1	
Cancer of œsophagus	3	
„ stomach	3	
„ sigmoid flexure	1	
„ bile duct	1	
Rupture of œsophagus	1	
Gastric ulcer	1	
Volvulus	1	
Colitis	1	
Cirrhosis	2	
Inpaction of gall stone	1	
Peritonitis	10	
Diarrhœa, infantile	11	
	—	37
Diseases of the Lymphatic System:		
Cancer of the cervical glands	1	
Lymphadenoma	1	
	—	2
Diseases of the Urinary System:		
Chronic renal	5	
Miliary nephritis	1	
Hypertrophy of prostate	3	
Cystitis	1	
	—	10
Diseases of the Reproductive System:		
Cancer of breast	1	
Gangrene of uterus	1	
Cyst of ovary	1	
	—	3
Diseases of the Organs of Locomotion:		
Morbus coxæ	1	
Caries of spine	1	
Sarcoma of hip	1	
	—	3
Injuries and Surgical Operations:		
Burn	4	
Fracture of skull	4	
„ spine	2	
„ ribs	2	
„ arm	1	
Rupture of heart	1	

INJURIES AND SURGICAL OPERATIONS—*continued.*		
Rupture of liver	1	
Cellulitis	1	
Excision of vermiform appendix	1	
„ rectum	1	
Colotomy	1	
Hernia	7	
For bronchocele	1	
„ aneurysm	1	
„ intestinal obstruction	1	
Extravasation of urine	3	
Amputation of breast	1	
Hysterectomy	1	
	—	33
UNCLASSIFIED		2

Short notes on most of the foregoing cases. The numbers from 318—400 *refer to the folios in P. M. book, vol. v.; from* 1—194 *to those in vol. vi.*

GENERAL DISEASES.

Erysipelas.—163. Diffuse cellulitis of leg.

Diphtheria.—366. No membrane; faucial mucosa, tonsils, sub-maxillary and cervical glands much swollen; suppurative bronchitis. On admission gray membrane on both tonsils. Much albumen. 17. Membrane from epiglottis downwards, ending as a suppurative bronchitis; pneumonia; tubercles; tracheotomy. 47. Membrane from epiglottis downwards ending as a suppurative bronchitis; pneumonia; acute nephritis; tracheotomy. 146. Patches of membrane on tonsils; thick membrane from cords downwards ending as suppurative bronchitis; pneumonia; tracheotomy. 176. Membrane on soft palate; thick membrane from epiglottis downwards ending as suppurative bronchitis; pneumonia.

Septicæmia.—378. Age 7 weeks. Cerebro-spinal meningitis; pleuro-pneumonia; suppuration of joints and necrosis of bones. 31. Puerperal; uterus in condition of recent delivery; spondylolisthesis. 41. Diffuse cellulitis of leg; delirium tremens. 101. Old abscesses of liver; chronic otitis; numerous recent abscesses in brain. 119. Puerperal; laceration of vagina;

thrombosis of periuterine veins; thrombosis of superior longtitudinal sinus and congestion infarcts of brain; abscesses in lungs; endocarditis. 131. Abscesses and consolidation of lungs; suppuration of right wrist joint.

Delirium tremens.—128. Columnæ carneæ of heart show numerous petechiæ; these places on microscopical examination were found to be due to rupture of the vessels, causing destruction of the muscular tissue from the extravasation; probably one of the prominent causes of cardiac failure in alcoholic poisoning.

Rickets.—392. 61. Thymus greatly hypertrophied, multilobular.

Tuberculosis —319. Meningitis, and tubercles in lungs and liver. 326. Tubercles in lungs and kidneys; tubercular ulceration of gastric and intestinal mucosa; abdominal glands large and caseous. 328. Tubercular pneumonia, age 53. 356. Tubercular pneumonia, age 36. 374. Age 23. Meningitis. tubercles in lungs and kidneys. 385. Meningitis; tubercles in lungs, liver, spleen, kidneys, intestinal mucosa and abdominal glands. 399. Meningitis; caries of spine. 6. Tuberculosis of lungs and intestines; pleuro-pneumonia. 33. Caseation in brain; tubercles in lungs; abdominal glands and intestines. 34. Tuberculosis of lungs and intestines; adherent pericardium; caries of digital phalanges. 39. Tubercular caseation in brain; tubercles in liver and spleen; otitis. 51. Age 35. Acute tuberculosis of lungs; a few tubercles in kidneys. 59. Meningitis; tubercles in lungs and abdominal viscera. 81. Age 21. Meningitis; no tubercles except on piamater. 91. Tubercular caseations in brain; meningitis; tubercles in lungs and abdominal viscera; ulcers of stomach and intestines. 111. Tubercles in lungs only. 115. Tubercular caseations in brain and spinal cord; meningitis; tubercles in thoracic and abdominal viscera. 126. Tuberculosis of lungs and abdominal viscera. 132. Meningitis; simple pneumonia; tuberculosis of abdominal viscera. 178. Tubercular caseation in medulla; meningitis; tuberculosis of lungs and abdominal viscera; tubercular metritis and salpingitis; morbus coxæ. 183. Meningitis; otitis; tubercles in lungs and abdominal viscera.

Nervous System.

Cerebro-spinal Meningitis.—353. Suppurative meningitis of brain and cord; abscess of cerebellum; no osteitis. Age 4 months. 371. Suppurative meningitis; suppurative pleuritis; no osteitis. Age 40 years. 14. Age 45 years. Suppurative meningitis and softening of brain; suppurative pleuritis; chronic myocarditis; left wrist joint full of pus. 44. Otitis; suppurative meningitis. 85. Age 15. No osteitis or otitis; suppurative meningitis. 135. Suppurative meningitis; no otitis. 187. Suppurative meningitis; otitis.

Hydrocephalus.—355. Age 19. Ventricles and iter much distended; small caseous tumour on basilar artery; chronic meningitis. 190. Ventricles and iter much dilated, and contain a small quantity of pus. Pleuro-pneumonia; parovarian cyst.

Abscess.—375. Chronic abscess in left frontal lobe; acute cerebro-spinal meningitis; emphysema; chronic pleuritis and bronchitis.

Hæmorrhage.—320. Hæmorrhage into both crura-cerebri. Age 37. Heart and kidneys normal. 351. Destruction of right tempero-sphenoidal lobe and extravasation into ventricles; aneurysms of both vertebral arteries close to the basilar; kidneys not noteworthy. 93. Hæmorrhage into pons; crura-cerebri and medulla; hypertrophy of left ventricle of heart; contracting granular kidneys. Age 37 years.

Softening.—124. Yellow softening of distribution-area of left middle cerebral artery; descending sclerosis of spinal cord; calcareous nodule in large flap of mitral. 161. Yellow softening of distribution-area of right middle cerebral artery; calcareous deposit in mitral; coarse vegetations on aortic and mitral valves; atheroma of aorta.

Tumour.—159. Glioma growing from left basal ganglia and projecting into third ventricle. At the base it appears in the quadrilateral space.

Convulsions.—367, 86, 98, 109. "Infantile."

Ear.

Otitis.—3. Necrosis of petrous bone; cerebro-spinal meningitis and abscess in right tempero-sphenoidal lobe; chronic pleuritis; adherent pericardium; mitral and aortic valvulitis. 9. Necrosis of petrous bone; ulceration through dura into left lateral sinus, which is clotted up; suppurative pleuritis; abscesses in lungs.

Circulatory System.

Endocarditis.—323. Stenosis of mitral and tricuspid; recent vegetations on mitral, aortic, and tricuspid; viscera "cardiac"; stenosis of os uteri with dilatation of cervical canal. 334. Adherent pericardium; old and recent aortic valvulitis; left ventricle dilated and hypertrophied; chronic pleuritis; "fibroid" phthisis; tubercles in lungs; tubercular ulcerations in intestines; horseshoe kidney. 364. Mitral and tricuspid stenosis; viscera "cardiac"; stenosis of os uteri; parovarian cysts. 365. Chronic and recent aortic valvulitis; pneumonia of upper lobes. 384. Adherent pericardium; stenosis of mitral and tricuspid; aortic valvulitis; viscera cardiac. 386. Adherent pericardium; tricuspid, mitral, and aortic valvulitis; viscera cardiac. 13. Stenosis of mitral and tricuspid; recent vegetations on all four valves; atrophy of left kidney; compensating hypertrophy of right. 40. Mitral and tricuspid stenosis; recent vegetations on the pulmonary valve and atheroma of the pulmonary artery; viscera cardiac; stenosis of os uteri; parovarian cyst. 45. Partial adhesion of pericardium; mitral stenosis; viscera cardiac; yellow softening of island of Reil and adjacent tissue, and also of central half of superior surface of left cerebellum. 53. Old and recent pericarditis; chronic mitral and aortic valvulitis. 64. Adherent pericardium; old and recent mitral and aortic valvulitis; pleuro-pneumonia kidneys, 13½ and 14 oz.; 71. Aortic valvulitis; aortitis; fœtus in utero. 84. Chronic mitral and aortic valvulitis; recent vegetations on aortic cusps; adherent pericardium; viscera cardiac. 95. Mitral stenosis; viscera cardiac; subinvolution of uterus. 107. Chronic mitral and aortic valvulitis; recent endocarditis of left auricle; infarcts in spleen and kidneys; uterus 3⅜ inch in length, in position of moderate

anteflexion; os patulous; mucosa of fundus, soft, swollen and hyperæmic; surface covered with thin grey slough. 143. Chronic and recent aortic valvulitis; chronic (calcareous) aortitis; anterior coronary artery has a double opening; hydroceles of both tunicæ vaginales.

Aortitis.—24. Abdominal aneurysm rupturing into pleural sac and eroding lumbar vertibræ. 28. Thoracic aneurysm eating through wall of left thorax and bursting in subscapular region, and also eroding vertebral column; recent aortic valvulitis. 48. Thoracic aneurysm rupturing into pericardial sac; arteries generally very atheromatous; atrophy of cortex of central third of left kidney; no laminated fibrin in sac of aneurysm. 96. Considerable aortitis with aneurysmal dilatation above valve which is in the condition of chronic valvulitis; viscera cardiac; myoma uteri; chronic pelvic peritonitis and cellulitis. 121. Well marked aortitis of ascending and transverse portions of arch, with only moderate aortic valvulitis; viscera cardiac. 154. Aorta throughout in condition of soft atheroma, and showing in places aneurysmal bulgings, one of which is of considerable size; adherent pericardium; hypertrophy of left ventricle; kidneys small and granular.

Pericarditis.—70. Age 2 years; suppurative pericarditis; acute pleuritis; pneumonia; suppurative bronchitis. 136. Old and recent pericarditis; partially adherent pericardium; recent vegetations on mitral, aortic, and tricuspid. 188. Pyopericarditis; partially adherent pericardium; empyema, with much thickening of pleura; viscera cardiac. 192. Old and recent pericarditis; vegetations of mitral; hydrothorax; lungs carnified and pneumonic.

Congenital.—383. Interventricular septum defective at base, and the flaps of mitral and tricuspid are adherent to the margin of the orifice; congenital syphilis.

Respiration.

Acute bronchitis.—27. Brought in dead.

Broncho-pneumonia.—343, 380. Chronic pleuritis; fibrosis of lungs; considerable hypertrophy of right ventricle; several

large cysts in left kidney. Age 6 years. 394, 10, 12, 18, 37. Cadaveric softening of stomach and intestines. 38, 90. Most of the cartilage of the right shoulder joint is eroded, leaving exposed the subjacent bone. The joint capsule is distended from the presence of a thin but somewhat turbid fluid. 100, 147, 114, 139.

Pneumonia.—332. Grey hepatisation, both lungs; spleen large and soft. 346. Grey hepatisation, right lung; spleen 5½ oz., very soft. 347. Grey hepatisation, right lung; spleen 8¾ oz., firm and dark; kidneys 3¼ and 4½ oz.; surface smooth, cortex large; colour whitish-yellow, mottled with irregular areas of red purple. Microscopical examination showed an interstitial nephritis, the malpighians being much fibrosed, but the arterioles not materially hypertrophied. 348. Grey hepatisation of right lung; red hepatisation of left; gall bladder choked with large calculus; subinvolution of uterus and endometritis; spleen 3 oz., soft and flabby. 354. Grey hepatisation of right lung; well marked substantive emphysema; spleen 5½ oz., pale and flabby. 372. Grey hepatisation, both lungs; spleen 9¼ oz., soft and pale. 373. Grey hepatisation of right lung; thickening of left pleura and consolidation of left lower lobe, a condition probably due to unresolved pneumonia; spleen 6¼ oz., pale and flabby. 8. Grey hepatisation, both lungs; spleen 7½ oz., pale and soft. 32. Grey hepatisation, right lung; spleen, 6¾ oz., pale and soft. 35. Red hepatisation, right lung. 56. Grey hepatisation. Both ventricles of heart are dilated, but not hypertrophied; viscera "cardiac"; spleen 3½ oz. 82. Grey hepatisation, right lung; spleen 4½ oz., soft and pale. 144. Grey hepatisation, right upper lobe; spleen 3½ oz. 145. Acute pleuritis of both sides. In right sac is large quantity of fluid by which the lung is compressed. The whole of the left lung is consolidated, and is of a dark red purple colour; spleen 1¾ oz. Age 1 year and 10 months.

Gangrene of lung.—400. Gangrene of right lung after pneumonia; pyopneumo-thorax; "pinhole" os uteri; chronic periovaritis.

Chronic bronchitis and emphysema.—322, 329, 330, 345. Lungs studded with black fibroid nodules, which on microsco-

pical examination were found to consist of aggregations of alveoli blocked with cells and fibrin. 360, 16. Chronic peritonitis. In mesenteric glands and in intestinal muscosa numerous calcareous nodules were found, and these on microscopical examination were found to consist of three zones, the outer composed of concentric layers of fibrous tissue, the middle of connective tissue and blood-vessels, while the centre or nucleus is an aggregation of calcareous granules. No remains of a parasite were detected. 80, 138. Projecting into the cavity of the right auricle, but quite covered with endocardium, is a spheroidal calcification of an amber colour, semi-transparent and brittle. It is about the size of a small pea, and resembles a little pebble. Left oblique inguinal hernia.

Phthisis.—318. Chronic pleuritis; fibroid and pneumonic phthisis with tubercles and vomicæ; tubercular ulceration of colon. 344. Chronic and recent pleuritis; tubercular pneumonic phthisis; vomicæ; ulceration of vocal cords; one ulcer of ileum. 363. Chronic pleuritis; tubercular pneumonic phthisis; vomicæ; ulceration of intestines. 368. Chronic pleuritis; fibroid and pneumonic phthisis; caseation and cavities; ulceration of larynx; ulcers of intestines; chronic peritonitis. 370. Chronic pleuritis; fibroid and pneumonic phthisis; large cavities filled with recent blood clot; chronic pelvic peritonitis. 379. Chronic pleuritis; tubercular pneumonic phthisis; numerous small cavities; peritonitis; tubercular ulceration of intestines and of larynx; thrombosis of veins of the left lower extremity. 395. Chronic pleuritis; right pneumo-thorax, the right lung being collapsed and the pleural sac full of air and pus; fibroid tubercular pneumonic phthisis; cavities; tubercular ulcerations of intestines; left half of lower jaw absent, having been removed by surgical operation. Numerous fibrous scars on surface of liver and kidneys. 60. Chronic pleuritis; tubercular pneumonic phthisis; vomicæ. 67. Chronic pleuritis, with effusion into right pleural sac; fibroid tubercular phthisis; cavities. 75. Chronic pleuritis; tubercular pneumonic phthisis; cavities; tubercular ulceration of intestines. 83. Chronic pleuritis; tubercular pneumonic phthisis; ulceration of larynx. 112. Chronic pleuritis (slight). Both lungs almost completely consolidated, being in condition of acute tubercular pneumonic

phthisis. A few small cavities. The first six to eight inches of colon are covered by a thick whitish-yellow membrane; this in places strips off easily; but in other parts it is adherent, and the subjacent mucosa is rough and hyperæmic (pellicular colitis). There is no tubercular ulceration of the gastro-intestinal mucosa. 122. Chronic pleuritis; fibroid tubercular phthisis; vomicæ; tubercular ulceration of palate, larynx, and intestines. 123. Chronic pleuritis; tubercular pneumonic phthisis; cavities; tubercular ulceration of larynx, and of intestines. 194. Chronic pleuritis; tubercular pneumonic phthisis; vomicæ; tubercular ulceration of intestines.

Empyema.—117. Chronic pleuritis. On the right side pleura is enormously thickened, with small cavity leading by a fistulous passage behind diaphragm to the abdomen and pelvis, where there are not only large masses of fibrous tissue but also collections of pus; encysted empyema of left pleural sac. The lower lobes of both lungs are compressed, but in other respects normal; chronic mitral and aortic valvulitis; spleen and liver amyloid; kidneys, large white amyloid. 160. Age 11 weeks. Pus in left thorax; lung collapsed. 171. Empyema cavity at base of right thorax. Surgical wound communicates both with the empyema sac and also with an abscess of the liver; liver, spleen, and kidneys amyloid.

Digestion.

Epithelioma of lip.—184. Emphysema; bronchitis; adipose heart; granular kidneys; atheroma of arteries at base of brain. No operation.

Cancer of œsophagus.—333. Cancerous stricture of œsophagus eating into bronchus, and causing septic pneumonia of right lung. 19. Cancerous stricture of œsophagus at level of cricoid. The growth was an epithelioma, it invaded the thyroid body, and there were secondary deposits in lungs and liver. 76. Cancerous growth at level of pharyngo-œsophageal junction, causing stricture of œsophagus and necrosis of thyroid cartilage. The right recurrent laryngeal nerve was involved in the mass; tubercular pneumonia phthisis; tubercular ulcer in ileum; cicatrix of gastric ulcer about the centre of the lesser curvature.

Cancer of stomach.—43. Scirrho-encephaloid of pyloric end. 105. Scirrho-encephaloid of cardiac end; blocking œsophageal aperture. Cancer is fungating and invading left thorax, where it has set up acute pleuritis; œsophagus dilated and hypertrophied; acute and chronic peritonitis. Both lungs contain numerous black fibroid quiescent tubercles. 181. Scirrho-encephaloid; fungating at cardiac end where it has ruptured, setting up acute peritonitis. Secondary deposits in lung.

Cancer of sigmoid flexure.—137. Cancerous mass occluding gut and setting up peritonitis. On microscopical examination it is found to be a columnar epithelioma.

Cancer of bile duct.—130. Scirrhous mass involving bile duct; secondary deposits in liver and kidney.

Rupture of œsophagus.—21. Age 2 years. Lower half of œsophagus is almost entirely destroyed, possibly as the result of a digestive action after rupture. With the exception of suppurative bronchitis passing in places into pneumonia there were no other noteworthy morbid appearances.

Gastric ulcer.—167. Punched out ulcer about centre of lesser curvature. In its centre stands up the gaping end of a largish artery; stomach full of blood; chronic aortic and mitral valvulitis; atheroma of aorta; hypertrophy of left ventricle; kidneys granular, contracted, cystic.

Volvulus.—381. Ascending and transverse colon have made a double turn, the caput coli lying in left iliac fossa. The mesocolon is very large. The gut involved is greenish in colour and much distended, the intestine above and below is quite normal.

Colitis.—63. Brought in dead. Body much emaciated. Almost no contents of intestine. Mucosa of large intestine much thickened and ulcerated.

Cirrhosis.—11. Peritonitis. 23. Ulceration of larynx; umbilical hernia; erysipelas; kidneys, 15¾ and 16¼ oz.

Impacted gall stone.—66. Jaundice; recent pleuritis and peritonitis; mitral valvulitis; abscesses of liver; cystic dilatation of pancreas; gall stone in common bile duct.

Peritonitis.—321. Ulceration of vermiform appendix, the entrance to gut being quite occluded. 327. Ulcer of the duodenum. 336. Tubercular and simple suppurative peritonitis; tuberculosis of lungs; tubercular salpingitis. 350. Perforation of jejunum; ulcerations of gastric mucosa; chronic pneumonia of both lower lobes. 359. Ulceration of the vermiform appendix; fæcal calculoid. 30. Tubercular. 133. Ulceration of vermiform appendix; suppurative pleuritis; ulceration of diaphragm; pneumonia. 180. Jaundice; ascites; peritoneal and subperitoneal tissue everywhere very thick, whereby the calibre of gut is much reduced and the coats stiffened. Much fibrous tissue about the portal fissure. 87. Colloid ovarian cyst with numerous soft adhesions; abdominal section was attempted. 94. Ulceration of the vermiform appendix; chronic aortic valvulitis.

Diarrhœa.—349, 382, 78, 88, 99, 113, 116, 129, 142, 148, 149.

Lymphatic System.

Cancer of the cervical glands.—388. Epithelioma.

Lymphadenoma.—390. Lymphatic glands, especially those of thorax, much enlarged. Spleen very large with numerous yellow toughish areas.

Urinary System.

Chronic renal.—5. Kidneys large, red, granular, āā 7 oz.; hypertrophy of left ventricle. 36. Age 17. Amputation at hip joint 10 years before; kidneys large, white; no reaction to iodine; effusion into pleural sac; pneumonia. 97. Both kidneys very irregular in shape, surface coarsely granular; cortex diminished; hypertrophy of left ventricle. 162. Small, red, granular; chronic endarteritis; yellow softening of pons varolii. 177. Large white; gastric ulcers on lesser curvature; hypertrophy of left ventricle.

Miliary nephritis.—157. Numerous miliary abscesses in left kidney. Bladder contains large quantity of turbid urine, which on examination was found to be due to pus cells. No evidence of cystitis; right kidney normal; chronic aortic valvulitis.

Hypertrophy of prostate.—325. Cystitis; miliary abscesses in kidneys; chronic valvulitis and aortitis; emphysema; bronchitis. 62. Cystitis; abscesses in kidneys; chronic endarteritis; yellow softening of left corpus striatum. 151. Bronchitis; emphysema; cystitis.

Reproductive System.

Cancer of Breast.—174. Scirrhus of left mamma; secondary deposits in brain, lungs, liver, lymphatic glands, and bones; uterus large; right ovary quite rudimentary.

Gangrene of uterus.—397. Uterus in condition of recent delivery; sloughing of posterior wall of fundus; peritonitis.

Cyst of Ovary.—152. Ascites; hydro-thorax; large thick-walled cyst of left ovary; chronic peritonitis; one kidney granular; omental umbilical hernia.

Organs of Locomotion.

Caries of spine.—58. Angular curvature of cervico-dorsal region. Both pleuræ much thickened, much fibrous tissue in left upper lobe; bronchitis. No tuberculosis or caseation anywhere.

Morbus coxæ.—127. Viscera amyloid; chronic aortic valvulitis. No evidence of tubercle in thorax or abdomen.

Sarcoma of Hip.—389. Growing from the fibrous tissue elements about the left hip joint. Secondary deposits in prevertebral glands, in kidneys and adrenals.

Injuries and Surgical Operations.

Burn.—79. Very great hyperæmia of skin and subcutaneous tissues, of intestines, and of cerebral cortex. 170, 173, 166.

Fracture of skull.—357. Of the base, with laceration of brain. 377. Of the vertex; suppurative cerebro-spinal meningitis; pneumonia. 42. Of the occipital region, with considerable hæmorrhage into brain; old united fracture of tibia and fibula; chronic

aortic valvulitis. 150. Of the temporo-parietal region; large subdural hæmorrhage causing considerable depression of cortex. Several hæmorrhages into brain; chronic endarteritis; chronic mitral and aortic valvulitis; right oblique inguinal hernia.

Fracture of spine.—358. Cervical region; spinal cord much lacerated. 185. Cervical region (Atlas); also fracture of radius and ulna, of ribs; dislocation of humerus; rupture of heart and lung.

Fracture of ribs.—1. Laceration of lung, spleen and kidney; chronic pleuritis; adherent pericardium; left inguinal omental hernia. 341. Laceration of lung.

Fracture of arm.—74. Fracture of ribs.

Rupture of heart.—361. Spontanecus; left ventricle.

Rupture of liver.—57.

Cellulitis. 338. Of leg; double pneumonia.

Excision of vermiform appendix.—52. chronic pleuritis; tuberculosis of lungs; chronic peritonitis, with large masses of fibrous tissue in upper pelvis, and in lumbar region.

Excision of rectum.—387. For cancer; diffuse suppuration of pelvic connective tissue.

Colotomy.—168. For malignant stricture of descending colon; peritonitis; acute mitral valvulitis.

Hernia.—324. Femoral; suppurative parotitis; chronic mitral and aortic valvulitis. 331. Strangulated umbilical; brought in dead. Uterine myomata; cysts in both ovaries. 339. Double femoral; knuckle of ilium gangrenous and adherent to right femoral ring. Left side shows scar of operation wound for strangulation some years ago; fibroid phthisis. 362. femoral; surgical wound opens into ileum. Uterus large; pelvis filled with mass of thick fibrous tissue, in middle of which is ragged cavity filled with foul-smelling pus. This cavity communicates with bladder by a fistulous aperture. Miliary abscesses of right kidney. 73. Femoral; artificial anus; adherent pericardium; mitral and aortic valvulitis; myomata uteri. 89.

Age 1 year 10 months; inguinal; peritonitis. 141. Femoral; peritonitis; chronic mitral and tricuspid valvulitis; os uteri "pinhole"; pus distending cavity of uterus and fallopian tubes.

Bronchocele.—155. Tracheotomy; fibro-celloid enlargement thyroid.

Aneurysm.—393. Subclavian fibroid phthisis; aortitis; left scrotal hernia.

Intestinal obstruction.—103. Chronic peritonitis, by which omentum had formed a bridge, under which a knuckle of gut had slipped and become incarcerated; resection of knuckle of gut; acute peritonitis.

Extravasation of urine.—26. Cystitis, large prostate; no stricture. 165. Stricture of urethra; perinæal fistulæ. 193. No stricture; bladder hypertrophied; prostate large; old abscess by side of urethra.

Amputation of breast.—164. Suppurative pleuritis and pericarditis; chronic peritonitis; parovarian cyst.

Hysterectomy.—54. For enormous fibro-myoma; laceration of bladder.

Unclassified.—68. This case is fully reported in vol. 6 of the W. H. Reports, page 129, and is denominated a case of fever of undetermined origin. 140. Age 18. Admitted for diarrhœa, vomiting, and abdominal pains. Much albumen in urine. Temperature normal. Taken ill September 27th. Died October 1st. Shortly before death temperature rose to 105°. P. M., nothing strikingly abnormal. Viscera "febrile." Microscopical examination of kidney; a few fibrosed malpighians. Throughout the sections show signs of great activity, the epithelia being swollen and granular, and about the arterioles and capillaries there is marked excess of nuclei and lucocytes.

MEDICAL REGISTRAR'S REPORT FOR THE YEAR 1890.

By Dr. WILLS.

Number of patients in Hospital, Jan. 1st, 1890			80
" " " Dec. 31st, 1890			72
Admitted to Medical Wards during the year			1,200
Of these—Males	659		
Females	541		
Average number of patients in Hospital			82·67
Average stay in Hospital of each patient			25·19
Recovered or relieved			981
Males	533		
Females	448		
Unrelieved, including those sent to Surgical or Obstetrical Wards			50
Males	24		
Females	26		
Died			169
Males	102		
Females	67		

The mortality during the year was 13·2 per cent.

Table A.

DISEASE.	Jan. to March.	April to June.	July to Sept.	Oct. to Dec.	Sex. Males.	Sex. Females.	Sex. Total.	Age. Under 5 yrs.	Age. 5—10.	Age. 10—20.	Age. 20—30.	Age. 30—40.	Age. 40—50.	Age. 50—60.	Age. Over 60.	Result. Death.	Result. Unrelieved.	Result. Relieved.	Remarks.
General Diseases:																			
Group A:																			
Scarlatina	1	3	0	2	...	6	6	...	1	4	1	...	...	...	...	...	5	1	
Morbilli	...	3	4	1	4	4	8	6	1	1	...	...	...	...	...	2	1	5	
Enteric fever	4	2	5	7	4	14	18	...	5	7	4	2	...	...	...	1	...	17	
Diphtheria	5	3	...	4	3	9	12	10	2	...	...	...	...	...	...	8	...	4	
Febricula	2	2	2	1	1	6	7	1	2	1	2	1	...	...	...	...	...	7	
Influenza	23	...	1	1	12	13	25	1	...	8	11	4	...	1	...	...	1	24	
Erysipelas	3	2	...	1	5	1	6	...	...	1	...	2	1	1	1	1	...	5	
Pertussis	...	3	4	...	4	3	7	5	2	...	...	...	...	...	...	1	...	6	
Intermittent fever	1	1	...	...	2	...	2	...	...	...	1	1	...	...	...	...	...	2	
Pyæmia	...	1	...	...	1	...	1	...	...	...	...	...	1	...	...	1	...	...	
Parotitis	...	...	...	1	1	...	1	...	...	1	...	...	...	...	...	...	...	1	
Syphilis:																			
1. Congenital	3	1	...	...	1	3	4	4	...	...	...	...	...	...	...	1	...	3	
2. Acquired	1	...	1	...	1	1	2	...	...	...	2	...	...	...	...	...	...	2	
Gonorrhœa	...	...	...	1	1	...	1	...	...	...	1	...	...	...	...	...	1	...	
Group B:																			
Poisons:																			
Alcohol	1	...	1	2	1	3	4	...	...	...	...	3	1	...	...	...	...	4	
Lead	...	...	3	1	4	...	4	...	...	...	2	1	1	...	...	...	...	4	
Group C:																			
Developmental Diseases:																			
Debility	...	1	...	...	1	...	1	...	...	...	1	...	...	...	...	...	...	1	
Group D:																			
Not classified:																			
Acute Rheumatism	9	6	21	23	35	24	59	1	3	26	19	6	3	1	...	2	1	56	
Subacute ditto	6	6	9	4	10	15	25	...	1	5	10	4	1	4	...	...	...	25	
Chronic ditto	2	4	5	...	2	9	11	...	...	...	...	1	3	5	2	...	...	11	
Gout	2	3	1	1	5	2	7	...	...	...	1	3	2	...	1	...	...	7	
Tuberculosis	5	11	3	1	11	9	20	12	3	1	2	2	...	...	...	15	3	2	
Rickets	2	4	1	2	5	4	9	9	...	...	...	...	...	...	...	...	...	9	
Myxœdema	1	...	...	...	...	1	1	...	...	...	...	1	...	...	...	...	...	1	
Anæmia	12	3	4	4	...	23	23	...	...	13	9	1	...	...	...	...	...	23	
Pernicious anæmia	...	...	1	...	1	...	1	...	...	...	...	1	...	...	...	...	...	1	
Hodgkin's disease	1	...	...	...	1	...	1	...	...	1	...	...	...	...	...	1	...	...	
Leucocythæmia	2	...	...	1	2	1	3	1	...	...	...	...	1	1	...	...	...	3	
Diabetes	...	1	...	1	1	1	2	...	...	...	...	1	1	...	...	...	1	1	
Diseases of the Nervous System:																			
Brain:																			
Hæmorrhage	1	1	...	...	1	1	2	...	...	...	...	...	...	2	...	1	1	...	
Hemiplegia	5	3	...	1	7	2	9	...	...	1	2	...	2	2	2	1	...	8	

TABLE A—*continued.*

DISEASE.	Jan. to March.	April to June.	July to Sept.	Oct. to Dec.	Sex. Males.	Females.	Total.	Age. Under 5 yrs.	5—10.	10—20.	20—30.	30—40.	40—50.	50—60.	Over 60.	Result. Death.	Unrelieved.	Relieved.	Remarks.
DISEASES OF THE NERVOUS SYSTEM—*continued*—																			
Tumour	3	2	1	2	6	2	8	...	2	1	1	2	2	...	...	2	1	5	
Abscess	1	...	...	...	1	...	1	...	...	...	...	...	...	1	...	1	...	...	
Hydrocephalus	1	...	...	2	...	3	3	2	...	...	...	...	1	...	...	1	1	1	
Meningitis	2	1	4	1	6	2	8	4	...	4	...	...	...	...	...	6	...	2	
Cerebro-spinal sclerosis	...	...	1	...	1	...	1	...	...	...	...	1	...	...	...	...	1	...	
Thrombosis of lateral sinus	...	1	...	...	...	1	1	...	...	1	...	...	...	...	...	1	...	...	
Cord:																			
Anterior polio-myelitis	1	...	...	...	1	...	1	1	...	...	...	...	...	...	...	...	1	...	
Spastic paralysis	1	...	...	...	1	...	1	...	...	1	...	...	...	...	...	...	...	1	
Tabes dorsalis	3	3	...	...	6	...	6	...	...	...	...	...	4	2	...	...	...	6	
Progressive muscular atrophy	...	...	1	...	...	1	1	...	...	...	...	1	...	...	...	...	...	1	
Paraplegia from Pott's disease	...	1	...	...	...	1	1	...	...	1	...	...	...	...	...	...	...	1	
Syphilitic disease	...	1	...	...	1	...	1	...	...	...	...	...	1	...	...	...	...	1	
Various:																			
Sciatica	2	3	...	4	8	1	9	...	...	...	1	1	3	2	2	...	...	9	
Alcoholic peripheral neuritis	4	...	...	...	2	2	4	...	...	...	1	1	1	...	1	...	...	4	
Diphtheritic paralysis	3	...	1	...	3	1	4	2	2	...	...	...	...	...	...	...	...	4	
Laryngismus stridulus	1	...	...	...	1	...	1	1	...	...	...	...	...	...	...	1	...	...	
Epilepsy	...	1	3	4	6	2	8	...	...	4	...	...	1	2	1	...	...	8	
Infantile convulsions	2	...	...	...	1	1	2	2	...	...	...	...	...	...	...	...	...	2	
Chorea	9	3	2	5	5	14	19	...	4	13	2	...	...	...	...	...	...	19	
Hysteria	8	4	5	2	5	14	19	...	...	2	11	4	2	...	...	...	...	19	
Hystero-epilepsy	...	1	...	...	1	...	1	...	...	...	...	...	...	1	...	...	...	1	
Paralysis agitans	1	...	...	...	1	...	1	...	...	...	...	...	...	...	1	...	...	1	
Facial neuralgia	1	...	...	...	...	1	1	...	...	1	...	...	...	...	...	...	...	1	
Vertigo	...	1	...	...	1	...	1	...	...	...	...	...	1	...	...	...	...	1	
Traumatic neuritis	...	...	1	...	1	...	1	...	...	...	...	...	1	...	...	...	...	1	
Spasmodic torticollis	...	1	...	...	1	...	1	...	...	...	...	...	...	1	...	...	...	1	
Shock	...	...	...	2	2	...	2	...	...	1	1	...	...	...	...	...	...	2	
Tetany	...	1	...	...	1	...	...	1	...	...	...	...	...	...	...	...	...	1	
Mental:																			
Idiocy	1	...	...	...	1	...	1	...	1	...	...	...	...	...	...	...	1	...	
General paralysis	3	1	1	...	3	2	5	...	...	...	...	2	3	...	...	...	4	1	
Melancholia	1	...	...	...	1	...	1	...	...	...	...	1	...	...	...	...	...	1	
Delirium tremens	...	...	2	...	1	1	2	...	...	...	...	2	...	...	...	1	...	1	
DISEASES OF THE CIRCULATORY SYSTEM:																			
Dilatation of heart	...	1	1	...	...	2	2	...	...	...	...	...	1	1	...	...	...	2	

TABLE A—*continued.*

DISEASE.	Jan. to March.	April to June.	July to Sept.	Oct. to Dec.	Sex.			Age.							Result.			Remarks.
					Males.	Females.	Total.	Under 5 yrs.	5—10.	10—20.	20—30.	30—40.	40—50.	50—60.	Over 60.	Death.	Unrelieved.	Relieved.	
DISEASES OF THE CIRCULATORY SYSTEM—*continued*—																		
Endocarditis (not arising in hospital) :																		
Mitral :																		
1. With presystolic murmur	1	...	4	...	2	3	5	...	...	1	...	1	3	...	...	...	...	5	
2. With systolic murmur	5	3	8	4	9	11	20	...	1	5	4	1	7	1	1	...	1	19	
3. With double murmur	15	8	6	6	11	24	35	...	1	12	6	10	5	...	...	11	1	23	
Aortic :																			
1. With systolic murmur	...	...	1	...	1	...	1	...	...	1	...	...	...	...	...	...	...	1	
2. With double murmur	9	4	4	6	15	8	23	...	...	6	3	3	3	6	2	3	2	18	
Ulcerative	...	...	1	...	1	...	1	...	...	...	...	...	...	1	...	1	...	...	
Pericarditis	1	3	1	...	4	1	5	1	...	...	3	...	...	1	...	1	...	4	
Cyanosis	...	1	...	...	1	...	1	...	...	...	1	...	...	...	...	...	...	1	
Angina pectoris	1	...	...	...	1	...	1	...	...	...	...	1	...	...	...	...	...	1	
Aneurysm thoracic	5	2	1	1	8	1	9	...	...	...	...	3	3	2	1	5	...	4	
„ abdominal	...	1	1	3	4	1	5	...	...	...	1	1	1	1	1	1	1	3	
Phlegmasia dolens	...	1	...	1	1	1	2	...	...	1	...	...	1	...	...	...	...	2	
DISEASES OF THE RESPIRATORY SYSTEM :																		
Rhinitis vasomotoria	1	...	...	...	1	...	1	...	...	...	...	1	...	...	...	...	...	1	
Acute laryngitis	1	1	2	4	5	3	8	1	1	2	3	...	...	1	...	...	...	8	
Bronchitis :																			
1. Acute	12	3	4	7	12	14	26	17	6	1	1	1	...	...	...	2	...	24	
2. Chronic	22	7	9	5	24	19	43	2	1	2	5	11	11	5	6	2	2	40	
Bronchiectasis	2	...	...	...	2	...	2	...	2	...	...	...	...	...	...	...	...	2	
Pneumonia :																			
1. Lobar	23	22	20	14	53	26	79	13	11	10	18	14	7	3	3	16	...	63	
2. Lobular	10	11	10	4	22	13	35	30	4	...	...	...	1	...	...	10	...	25	
Gangrene	...	1	...	...	...	1	1	...	...	...	...	1	...	...	...	1	...	...	
Phthisis	36	18	24	9	60	27	87	...	2	21	24	14	14	5	7	23	4	60	
Malignant disease of lung	1	1	1	...	2	1	3	...	...	...	...	2	...	1	...	...	1	2	
Emphysema	3	5	2	1	11	...	11	...	1	...	2	2	1	3	2	4	...	7	
Pleurisy :																			
1. Without effusion	10	5	2	1	12	6	18	1	2	2	7	4	2	...	...	...	...	18	
2. With effusion	9	10	7	3	24	5	29	2	4	13	2	6	...	2	...	1	...	28	
3. Empyema	6	...	2	2	5	5	10	4	3	...	...	...	1	1	1	3	1	6	
Pneumo thorax	1	...	...	...	1	...	1	...	...	...	...	...	1	...	...	...	...	1	
Asthma	...	...	1	...	1	...	1	...	...	...	1	...	...	...	...	...	...	1	

TABLE A—*continued.*

DISEASE.	Jan. to March.	April to June.	July to Sept.	Oct. to Dec.	Sex. Males.	Sex. Females.	Sex. Total.	Age. Under 5 yrs.	Age. 5—10.	Age. 10—20.	Age. 20—30.	Age. 30—40.	Age. 40—50	Age. 50—60.	Age. Over 60.	Result. Death.	Result. Unrelieved.	Result. Relieved.	Remarks.
DISEASES OF THE DIGESTIVE SYSTEM:																			
Mouth:																			
Stomatitis	1	...	...	...	...	1	1	...	...	1	...	...	...	...	...	...	...	1	
Pharynx and Œsophagus:																			
Tonsillitis	9	12	15	13	31	18	49	9	8	19	10	2	1	...	...	1	...	48	
Rupture of œsophagus	..	1	...	...	1	...	1	1	...	...	...	...	...	...	...	1	...	...	
Malignant disease of œsophagus	...	2	...	...	...	2	2	...	...	...	1	...	...	1	...	1	...	1	
Stomach:																			
Dyspepsia	2	1	2	3	2	6	8	...	...	...	3	2	1	2	...	...	...	8	
Acute gastritis	1	...	5	...	2	4	6	...	...	1	4	1	...	...	...	...	...	6	
Ulcer	9	4	4	8	5	20	25	...	...	5	11	6	1	1	1	2	4	19	
Carcinoma	3	4	2	1	4	6	10	...	...	...	...	...	4	5	1	3	1	6	
Dilatation	1	...	...	...	1	...	1	...	...	1	...	...	...	...	...	...	...	1	
Intestines:																			
Vomiting	1	2	5	1	2	7	9	2	3	3	...	1	...	...	...	...	...	9	
Diarrhœa	3	4	23	7	17	20	37	25	2	3	2	2	1	1	1	4	...	33	
Constipation	4	...	...	...	1	3	4	1	...	...	...	2	1	...	...	...	...	4	
Colic	2	2	8	4	12	4	16	1	2	5	3	3	1	1	...	...	...	16	
Typhlitis	1	2	1	2	5	1	6	...	1	3	1	1	...	...	...	...	...	6	
Enteritis	1	...	1	...	...	2	2	...	...	1	...	...	...	1	...	...	...	2	
Duodenal ulcer	2	1	...	1	4	...	4	...	...	...	1	1	...	2	...	1	...	3	
Perforation of Vermiform Appendix	1	...	2	...	3	...	3	...	1	...	1	...	...	1	...	2	...	1	
Atony of the intestines	...	...	1	...	1	...	1	...	...	...	...	...	...	1	...	...	...	1	
Stricture	...	...	1	...	...	1	1	...	...	1	...	...	...	...	...	1	...	...	
Malignant disease	...	...	2	1	1	2	3	...	...	...	...	...	...	3	...	...	1	2	
Melæna	...	...	1	...	1	...	1	...	...	...	...	...	1	...	...	...	...	1	
Tænia mediocanellata	...	...	1	...	1	...	1	...	1	...	...	...	...	...	...	...	...	1	
Liver:																			
Acute hepatitis	...	3	1	...	4	...	4	...	...	1	2	...	1	...	...	1	...	3	
Cirrhosis:																			
1. Atrophic	...	2	2	1	2	3	5	...	...	...	...	2	3	...	...	2	...	3	
2. Hypertrophic	2	...	1	...	3	...	3	...	...	...	...	2	1	...	...	...	...	3	
Malignant disease	...	1	1	1	2	1	3	...	...	...	...	...	1	2	...	1	...	2	
Syphilitic disease	1	...	...	...	1	...	1	...	...	...	...	1	...	...	...	...	...	1	
Gall stones	...	4	...	2	...	6	6	...	...	...	1	3	...	2	...	1	...	5	
Jaundice	3	...	2	...	3	2	5	...	...	...	2	1	1	...	1	...	...	5	
Peritoneum:																			
Peritonitis:																			
1. Acute	1	...	...	...	...	1	1	...	...	1	...	...	...	...	...	...	...	1	
2. Chronic	...	...	2	2	1	3	4	1	...	1	1	...	...	...	1	2	...	2	
Malignant disease	...	1	...	1	1	1	2	...	...	...	1	...	...	1	...	1	...	1	
Ascites	1	3	1	...	3	2	5	...	...	...	...	...	3	...	2	...	...	5	

TABLE A—*continued.*

DISEASE.	Jan. to March.	April to June.	July to Sept.	Oct. to Dec.	Sex. Males.	Sex. Females.	Sex. Total.	Age. Under 5 yrs.	Age. 5—10.	Age. 10—20.	Age. 20—30.	Age. 30—40.	Age. 40—50.	Age. 50—60.	Age. Over 60.	Result. Death.	Result. Unrelieved.	Result. Relieved.	Remarks.
DISEASES OF THE LYMPHATIC SYSTEM:																			
Adenitis	1	...	...	...	1	...	1	...	...	1	...	...	...	...	...	...	...	1	
DISEASES OF THE THYROID GLAND:																			
Exophthalmic goitre	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	
Malignant disease	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	...	
DISEASES OF THE URINARY SYSTEM:																			
Acute nephritis	3	...	1	...	4	...	4	2	...	...	1	...	1	...	...	...	...	4	
Chronic nephritis:																			
1. Large white kidney	2	9	9	3	16	7	23	...	1	7	3	7	4	...	1	3	2	18	
2. Contracted	5	3	3	2	7	6	13	...	...	1	2	...	4	5	1	3	1	9	
3. Suppurative	...	...	1	...	1	...	1	...	...	...	...	...	1	...	...	1	...	...	
Movable kidney	...	1	...	...	...	1	1	...	...	...	...	...	1	...	...	...	...	1	
Renal calculus	1	1	1	1	2	2	4	...	...	2	1	1	...	...	...	...	1	3	
Growth in bladder	1	...	...	...	...	1	1	...	...	...	...	...	...	1	...	...	...	1	
Urethral stricture	...	1	...	...	1	...	1	...	...	...	...	...	...	1	...	...	...	1	
Incontinence of urine	1	...	...	...	...	1	1	...	...	...	1	...	...	...	...	...	...	1	
Phimosis	1	...	...	...	1	...	1	...	...	1	...	...	...	...	...	...	...	1	
DISEASES OF THE SKIN:																			
Erythema nodosum	...	1	...	1	...	2	2	...	...	...	...	1	...	...	1	...	...	2	
Eczema	...	3	1	...	1	3	4	1	...	...	1	1	...	...	1	...	...	4	
Lupus	...	2	...	...	...	2	2	...	...	1	1	...	...	...	...	...	...	2	
Dermatitis herpetiformis	...	1	...	...	...	1	1	...	...	...	1	...	...	...	...	...	...	1	
Psoriasis	1	...	...	...	1	...	1	...	...	...	...	...	1	...	...	...	...	1	
DISEASES OF WOMEN:																			
Ovarian tumour	...	...	1	...	...	1	1	...	...	...	...	1	...	...	...	...	...	1	
Malignant disease of uterus	...	...	1	...	...	1	1	...	...	...	...	...	...	1	...	...	1	...	
Endocervicitis	1	...	...	...	...	1	1	...	...	...	1	...	...	...	...	...	...	1	
Pelvic peritonitis	...	...	1	...	...	1	1	...	...	...	...	1	...	...	...	...	...	1	
Pelvic hæmatocele	1	...	...	...	...	1	1	...	...	...	1	...	...	...	...	...	...	1	
Menorrhagia	...	...	1	...	...	1	1	...	...	...	...	1	...	...	...	...	...	1	
VARIOUS:																			
Marasmus	3	1	8	3	9	6	15	13	1	...	...	1	...	...	...	6	2	4	
Otitis media	2	1	1	...	3	1	4	2	1	...	1	...	...	...	...	...	1	3	
Lympho-sarcoma of neck	...	1	...	...	1	...	1	...	...	...	...	...	...	...	1	...	1	...	
Sarcoma of pelvis	1	1	...	...	2	...	2	...	...	...	...	...	...	2	...	1	1	...	
Inflammation of breast	...	...	1	...	...	1	1	...	...	...	...	1	...	...	...	...	...	1	
Nil	3	5	3	2	5	8	13	3	4	4	1	1	...	...	...	...	...	13	

Table B.—*Enteric Fever.*

Eighteen cases of Enteric Fever were treated in the hospital during the year 1890.

The average stay in the hospital of each patient was 52·72 days.

During the first four months of the year only four cases of Enteric were admitted against ten during the last four months.

The number of cases having constipation throughout their illness was only five this year, a decidedly smaller proportion than last year. Profuse diarrhœa occurred in only three cases. Five patients suffered relapse, two of them being admitted during the relapse, one of whom did not take to her bed in the original attack at all. In one case there was hæmorrhage from the bowel.

There was one death, probably from peritonitis after perforation; but no post-mortem examination was allowed.

In only two instances were two patients admitted from the same house.

The cause assigned was in four cases bad drainage. In one—a nurse—it seemed probable that direct infection from the boy she was in charge of had taken place; in the others, no cause could be ascertained.

TABLE B—*continued.*

No. and Name.	Month admitted.	Sex.	Age.	Onset.	Character.	Fever.	Days in hosp.	Result.	Treatment.	Remarks.
1 A. P.	Jan.	F.	12	Pains in head and stomach	Admitted on 8th day; abdomen tumid and tender; bowels constipated, then loose; tongue very dry and furred; bronchitis. Slow convalescence	18 days' fever; highest temp. 104°	56	Recov.	—	Came from the same house at Hendon as three other children admitted during 1889.
2 T. H.	„	M.	28	Anorexia. Morning vomiting	Admitted on 8th day; abdomen distended; spots; diarrhœa; very tremulous, and later on delirious. On 3rd day developed pneumonia at left apex, which followed a normal course	12 days' fever; highest temp.	29	„	Brandy, opium	Lived in the same court as A. J. (Table B., No. 10, 89). Slight albuminuria, which persisted throughout febrile period. It was doubtful whether this was a case of enteric or simply pneumonia.
3 J. G.	Feb.	F.	19	Tolerably sudden onset. Shivering, anorexia, diarrhœa	Admitted on the 8th day; abdomen slightly distended; spots; diarrhœa; spleen enlarged; tongue furred; slight bronchitis. Good convalescence. Phlegmasia alba of left leg came on 17th day after admission, and lasted about a week	17 days' fever; highest temp. 103·2°	43	„	Brandy	A mild case. Apparently admitted during relapse; First attack having come on 4 weeks previously.

4 H. Mc. K.	Mar.	F.	7	Wasting, anorexia, feverishness	Admitted on the 15th day; abdomen slightly distended; no spots; bowels constipated; slight bronchitis. Relapse on 26th day of illness; abdomen much distended; numerous spots on abdomen and legs; bronchitis; diarrhœa. Very ill for a week, then good convalescence	7 days' fever; highest temp. 101·8°. In relapse temp. rose suddenly to 104·2°. 17 days' fever; highest temp. 105·4°	66	„	Brandy, opium	The first part of the attack was not recognised as enteric, as she came in at the end of the attack; but the diagnosis was made evident by the relapse. In the interval of 6 days she had been up and having solid food.
5 F. S.	May	M.	21	Pains in limbs, anorexia, vomiting	Admitted on 6th day; prostrate; face flushed; abdomen distended; tender; very numerous spots on chest and abdomen; diarrhœa; bronchitis; systolic apex murmur. On 8th and 9th day had severe hæmoptysis, afterwards passed motions in bed; pulse dicrotic; very much prostrated, then gradual convalescence much retarded by numerous abscesses on back and neck	33 days' fever; (last 16 probably due to the abscesses); highest temp. 104·2°	50	„	„	Albuminuria for first 10 days. Three members of same house had lately been ill for a week with diarrhœa. This was called "Influenza."
6 F. G.	„	F.	7½	Lassitude, headache, epistaxis, very delirious at night	Admitted on 14th day; emaciated; abdomen tumid; slight diarrhœa; spots; bronchitis. Relapse on 33rd day of illness; bowels constipated; spots; abdomen tumid. Good convalescence	14 days' fever; highest temp. 104·4°. In relapse 14 days' fever; highest temp. 103·4°	55	„	Hst. Aeth. et Ammon.	

TABLE B—*continued.*

No and Name.	Month admitted.	Sex.	Age.	Onset.	Character.	Fever.	Days in hosp.	Result.	Treatment.	Remarks.
7 M. M.	July	F.	6	Ailing for some time, vomiting, diarrhœa	Admitted on 4th day; abdomen tumid; bowels constipated. Mild case	20 days' fever; highest temp. 102·6°	63	Recov.	—	
8 W. W.	Aug.	M.	10	Headache, lassitude	Admitted on 8th day; abdomen tumid; spleen enlarged; slight diarrhœa; spots 10th day; much bronchitis	16 days' fever; highest temp. 104°	45	„	Enemata	
9 C. R.	Sept.	M.	34	Stated to have had rheumatic fever 14 days before admission. Intense headache was the main early symptom	Admitted on 8th day; abdomen tumid, tender; spleen enlarged; diarrhœa; numerous spots on chest and abdomen on 14th day; later was tremulous and exceedingly prostrate, then gradual recovery, interrupted by orchitis and cystitis	44 days' fever; (last 14 days owing to orchitis); highest temp. 105°	85	„	Brandy, quinine	Diagnosis was doubtful at first. He was thought to be possibly suffering from meningitis.
10 E. B. G.	„	F.	30	Gradual; headache, nausea, vomiting, diarrhœa	Admitted on 9th day; epistaxis; prostrate; abdomen tumid; constipation at first, then diarrhœa; considerable hæmorrhage from bowels on two occasions on 20th day. Collapsed and very ill, but gradually improved from that time	26 days' fever; highest temp. 104·6°	48	„	Purgat enemata, sponging. Pil. plumb. c. opio sodii salicyl.	A nurse. She was nursing a boy with enteric at Lea, who had caught it in Germany, and had just come home. She had been on night duty with the patient for 3 weeks, and apparently became infected directly from the patient.

11 H. W.	„	F.	19	Sudden headache, pains in limbs, constipation	Admitted on 9th day; prostrate; abdomen tumid; spleen slightly enlarged; spots. On 16th day vomited; great pain in abdomen and distension; collapsed; temp. fell to 97°; continued vomiting and hiccough; face drawn and anxious. Gradually got weaker, and died on the 9th day after the perforation (?)	16 days' fever; highest temp. 107·2°	16	Death. No P. M. allowed	Sponging, peptonised meat juice and suppos.; sulphonal	Apparently a mild case at first. Her sister had contracted enteric at Southend, and returned home to London, where she remained. This patient's fever terminated 14 days before the patient H. W. became ill. She was not nursing her sister, and had not seen her since the commencement of her illness. The drains were recently approved by the sanitary inspector.
12 P. W.	Oct.	F.	24	Gradual lassitude, anorexia, headache	Admitted on 15th day; drowsy; deaf; abdomen distended; spots; considerable diarrhœa; bronchitis. Good convalescence	13 days' fever; highest temp. 104·4°	43	Recov.	Enema amyli c. opio	
13 C. F.	„	F.	9	Ill for a month before admission with headache and occasional sickness	Admitted on 7th day; severe headache; photophobia; abdomen tumid; constipation throughout, except 1 day's diarrhœa. Good convalescence	24 days' fever; temp. was raised during the last week from constipation; highest temp. 104·4°	53	„	Purgative enemata	Was thought to be a case of meningitis at first. Resisted feeding and refused to talk for the last 14 days of fever, and during this time was very deaf, for which no cause could be found in the ear.
14 E. C.	„	F.	17	Sudden shivering, vomiting, and severe headache	Admitted on 2nd day; severe headache and constipation at first, then abdomen gradually became tumid. Spots appeared on 7th day; slight bronchitis; constipation throughout. Good convalescence	24 days' fever; highest temp. 103·8°	50	„	—	Also was thought to be probably a case of meningitis at first.

TABLE B—*continued.*

No. and Name.	Month admitted.	Sex.	Age.	Onset.	Character.	Fever.	Days in hosp.	Result.	Treatment.	Remarks.
15 G. W.	Nov.	F.	11	Sudden drowsiness, headache and vomiting	Admitted on 4th day; fauces reddened and painful; abdomen tumid; diarrhœa; a few doubtful spots; bronchitis. Good but slow convalescence	16 days' fever; highest temp. 105°	66	Recov.	Purgative enemata, brandy	Discharged in 1891.
16 S. H.	„	F.	10	Sudden shivering, drowsiness, pain in abdomen	Admitted on the 6th day; abdomen tumid; spots; slight bronchitis; constipation throughout. In relapse on 30th day, which was much more severe; prostrate; diarrhœa; very irritable; numerous spots; bronchitis. Good convalescence	23 days' fever; highest temp. 104·2°. In relapse temp. rose in 2 days to 102·2°. 21 days fever; highest temp. 105°	76	„	Purgative enemata. Sponged once	One other child in same school in St. Thomas's with enteric. Drains at school have been recently taken up. Discharged in 1891.
17 E. J.	„	F.	13	Pains and weakness in legs, headache, anorexia, pain in abdomen	Admitted on 14th day; rather prostrate; abdomen tumid; constipation at first, bowels regular afterwards; slight bronchitis; slow convalescence	42 days' fever; temp. continued to oscillate during last fortnight owing to constipation; highest temp. 104·8°	65	„	Sponged, Burgundy	The attack here described was probably a relapse. On admission there was marked knee and ankle clonus. Discharged 1891. A second relapse took place on 59th day, lasting 21 days. No symptoms. Highest temp. 103·2

18 R. R.	Dec.	F.	28	Never well since confinement three months before. 6 weeks ago mammary abscess; 1 week ago got worse with vomiting, diarrhœa and night delirium	Admitted on 9th day; very prostrate; abdomen much distended; spots; profuse diarrhœa and much bronchitis; tongue dry and brown. Good convalescence	15 days' fever; highest temp. 104·4°	48	„	Brandy	Was admitted as a case of metritis into Arden Ward. Discharged in 1891.

TABLE C.—*Acute Rheumatism.*

In last year's Report a summary was given of all the cases of acute rheumatism treated in the Hospital during the years 1881-2-3-4-5-6-7-8-9, with special reference to the following points: sex, age, months, seasons of the year, duration of attack, heart complications, and inheritance.

To this summary are now added the details of the cases treated during the year 1890, the total number of cases being 668.

Ages.

	Under 5.	5—10.	10—20.	20—30.	30—40.	40—50.	50—60.	Over 60.
1881	0	2	31	22	23	5	1	0
1882	1	3	29	16	12	5	1	0
1883	0	1	18	28	14	8	2	1
1884	0	16	33	29	14	5	3	0
1885	0	1	26	21	12	1	1	1
1886	0	0	17	13	4	2	0	0
1887	0	2	29	24	10	3	1	0
1888	1	5	18	29	9	3	0	0
1889	0	1	15	25	7	5	0	0
1890	1	3	26	19	6	3	1	0
Total	3	34	242	226	111	40	10	2

Sex.

	Males.	Females.
1881	43	41
1882	25	42
1883	39	33
1884	46	54
1885	31	32
1886	13	23
1887	34	35
1888	38	27
1889	29	24
1890	35	24
Total	333	335

Numbers admitted in each Month.

January	64	July	40
February	59	August	51
March	57	September	75
April	46	October	75
May	43	November	50
June	47	December	61

Seasons.

Cases admitted, in each group of three months.

January to March	180	July to September.....	167
April to June	136	October to December...	185

Cases admitted, in two groups: six warmer and six colder months.

April to September ...	296	October to March ...	372

Average stay in Hospital—26·04 days.

Inheritance.

	Rheumatism.	Gout.	Neurosis.	Phthisis.
1881	26	7	3	11
1882	34	2	13	19
1883	32	6	9	9
1884	42	14	22	8
1885	17	7	22	18
1886	11	2	3	5
1887	28	0	8	14
1888	37	3	0	9
1889	18	1	6	19
1890	30	12	7	18
Total.....	275	54	93	130

Character.

Months.	No. of Cases.	Average stay in days.	No. of Cases with heart disease.	Character of heart disease.		No. of Cases admitted with old heart disease.
				Peri-carditis.	Endo-carditis.	
January	64	31·18	38	13	35	9
February	59	29·96	38	21	27	11
March	57	29·66	30	15	26	6
April	46	34·23	26	8	23	11
May	43	29·92	23	13	20	7
June	47	26·62	26	10	20	9
July.....	40	23·34	25	8	19	12
August	51	22·05	26	12	18	11
September	75	26·73	38	14	29	15
October	75	31·77	48	20	36	17
November	50	26·5	37	17	20	7
December	61	25·33	33	13	22	12

The whole number of cases with heart affection is 388; of these 164 suffered from pericarditis, and 295 from endocarditis. The number includes 127 cases with old heart disease, as shown by cardiac bruits, and 106 who have had both pericarditis and endocarditis. The number leaving Hospital with persistent bruits is 302.

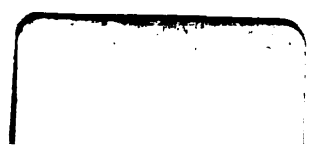

TABLE C.—*Acute Rheumatism.*

In last year's Report a summary was given of all the cases of acute rheumatism treated in the Hospital during the years 1881-2-3-4-5-6-7-8-9, with special reference to the following points: sex, age, months, seasons of the year, duration of attack, heart complications, and inheritance.

To this summary are now added the details of the cases treated during the year 1890, the total number of cases being 668.

Ages.

	Under 5.	5—10.	10—20.	20—30.	30—40.	40—50.	50—60.	Over 60.
1881	0	2	31	22	23	5	1	0
1882	1	3	29	16	12	5	1	0
1883	0	1	18	28	14	8	2	1
1884	0	16	33	29	14	5	3	0
1885	0	1	26	21	12	1	1	1
1886	0	0	17	13	4	2	0	0
1887	0	2	29	24	10	3	1	0
1888	1	5	18	29	9	3	0	0
1889	0	1	15	25	7	5	0	0
1890	1	3	26	19	6	3	1	0
Total	3	34	242	226	111	40	10	2

Sex.

	Males.	Females.
1881	43	41
1882	25	42
1883	39	33
1884	46	54
1885	31	32
1886	13	23
1887	34	35
1888	38	27
1889	29	24
1890	35	24
Total	333	335

Numbers admitted in each Month.

January	64	July	40
February	59	August	51
March	57	September	75
April	46	October	75
May	43	November	50
June	47	December	61

Seasons.

Cases admitted, in each group of three months.

January to March	180	July to September.....	167
April to June	136	October to December...	185

Cases admitted, in two groups: six warmer and six colder months.

April to September ...	296	October to March ...	372

Average stay in Hospital—26·04 days.

Inheritance.

	Rheumatism.	Gout.	Neurosis.	Phthisis.
1881	26	7	3	11
1882	34	2	13	19
1883	32	6	9	9
1884	42	14	22	8
1885	17	7	22	18
1886	11	2	3	5
1887	28	0	8	14
1888	37	3	0	9
1889	18	1	6	19
1890	30	12	7	18
Total.....	275	54	93	130

Character.

Months.	No. of Cases.	Average stay in days.	No. of Cases with heart disease.	Character of heart disease.		No. of Cases admitted with old heart disease.
				Peri-carditis.	Endo-carditis.	
January	64	31·18	38	13	35	9
February	59	29·96	38	21	27	11
March	57	29·66	30	15	26	6
April	46	34·23	26	8	23	11
May	43	29·92	23	13	20	7
June	47	26·62	26	10	20	9
July.....	40	23·34	25	8	19	12
August	51	22·05	26	12	18	11
September	75	26·73	38	14	29	15
October	75	31·77	48	20	36	17
November	50	26·5	37	17	20	7
December	61	25·33	33	13	22	12

The whole number of cases with heart affection is 388; of these 164 suffered from pericarditis, and 295 from endocarditis. The number includes 127 cases with old heart disease, as shown by cardiac bruits, and 106 who have had both pericarditis and endocarditis. The number leaving Hospital with persistent bruits is 302.

TABLE C—*continued.*

No. and Name.	Sex.	Age.	Month admitted	Day of illness admitted	Previous attacks.	Age when first attacked.	Family history.	Days of fever in hospital.	Days of pain in hospital.	Highest temp.	Joints, &c., affected.	Heart.			No. of days in hospital.	Result.	Treatment	Remarks.
												Affection.	In which attack developed.	State on discharge.				
1 G. D.	M.	20	Jan.	5th	0	20	Father has rheumatic gout	20	18	103°	Both knees, ankles, shoulders, wrists	Pericarditis, endocarditis	Present	Faint mitral systolic murmur, apex normal	44	Recovery	Hst. sod. sal.	Works in his linen shirt in the rain constantly. Became delirious on 3rd day, and continued so for 10 days and nights. No pneumonia. Epistaxis four times.
2 E. F.	F.	4	„	4th	0	4	Father has had rheumatism frequently, grandfather died from phthisis, 1 brother has hip disease	17	3	103·2°	Left hip, both knees, left ankle, fingers	Pericarditis, endocarditis	„	Mitral systolic murmur	44	„	—	Doubtful whether rheumatism or morbus coxæ on admission.
3 J. W.	M.	17	Feb.	3rd	0	17	Father has had acute rheumatism, maternal aunt insane, another has epilepsy	3	6	102°	Big toe, both knees	Functional apex systolic murmur	—	Heart sounds normal	13	„	Hst. sod. sal.	A plumber.

4 A. B.	F.	22	„	14th	1	15	Mother has rheumatism, 3 paternal uncles have gout	21	7	103°	Hips, knees, ankles	Functional apex systolic murmur	—	Heart sounds normal	26	„	Brandy	Heart symptoms predominated. Very anæmic.
5 W. L.	M.	16	„	6th	0	16	Father had acute rheumatism when young	4	8	102°	Knees, ankles	Pericarditis, endocarditis	Present	Mitral systolic murmur	27	„	Hst. pot. cit., pot. bicarb.	A temperate barman.
6 T. H.	M.	11	Mar.	5th	0	11	Maternal aunt has had acute rheumatism	17	7	103°	Left ankle, knees, wrists, elbows, right shoulder	Pericarditis, endocarditis	„	Very faint mitral systolic murmur, apex normal	37	„	Hydrag. subchlor.	Tonsillitis during attack.
7 L. H.	F.	26	„	7th	0	26	1 sister has had rheumatism, mother died of bronchitis, father died of rheumatic heart disease	3	4	100·8°	Right ankle and knee	Functional murmur at all orifices	—	Heart sounds normal	13	„	Hst. sod. sal.	Had a sore throat before illness. Feet got wet a few days before.
8 E. B.	M.	16	„	5th	0	16	—	21	26	102·8°	Roth ankles, shoulders, knees, wrists	Pericarditis, endocarditis	Present	Mitral and aortic (?) systolic murmurs	42	„	Hst. sod. sal., hst. pot. cit., pulv. ipecac. co.	1st cousin of H. D. (No. 10, Table C. 89.)

TABLE C—*continued.*

No. and Name.	Sex.	Age.	Month admitted	Day of illness admitted	Previous attacks.	Age when first attacked.	Family history.	Days of fever in hospital.	Days of pain in hospital.	Highest temp.	Joints, &c., affected.	Heart.			No. of days in hospital.	Result.	Treatment	Remarks.
												Affection.	In which attack developed.	State on discharge.				
9 S. B.	M.	20	Mar.	3rd	0	20	2 brothers have had acute rheumatism, 1 brother gout, paternal uncle insane	5	7	102·4°	Ankles, knees	—	—	—	29	Recovery	Hst. sod. sal.	A policeman. Suffering from syphilitic ulceration of throat on admission.
10 R. S.	M.	18	Apr.	6th	0	10	Father, mother, and 1 brother suffer from rheumatism	1	3	100°	Ankles, knees, right elbow very slight	Endocarditis	Present	Loud general systolic, especially at apex	19	„ Sent to fever hospital	—	Developed scarlet fever in hospital.
11 C. B.	F.	30	May	8th	1	15	Father has had acute rheumatism, mother died of heart disease	11	7	102°	Ankles, knees, elbows, wrists, hands	Endocarditis	„	Faint mitral systolic murmur	27	Recovery	Hst. sod. sal.	—

12 C. P.	M.	20	„	4th	4	12	Father and mother both have had rheumatism	42	30	102·8°	Ankles, knees, hips, wrists, elbows	Pericarditis, endocarditis	Previous and present	Double aortic and mitral systolic murmur, apex in 5th interspace outside nipple, probably adherent pericardium	92	„	Hst. sod. sal., ammon., brandy	Tonsillitis 1 week before acute rheumatism. Previous admission in 1888. (No. 61, Table C.) Lobar pneumonia and pleural effusion, first left, then right base. Aspirated three times. Heart and lung symptoms predominated.
13 H. M.	M.	23	June	4th	0	23	—	3	3	101·8°	Both knees, ankles, left shoulder	Functional systolic murmur at apex	—	Functional systolic murmur at apex	11	„	Hst. sod. sal.	Hæmaturia on admission, and lasting 3 days after.
14 J. H.	M.	39	„	9th	6	13	—	4	7	101·8°	Both knees, wrists	Endocarditis	Previous	Double mitral murmur, apex in nipple line	15	„	Hst. sod. sal., hst. pot. cit.	—
15 R. D.	M.	6¾	„	11th	0	6¾	—	6	2	101·6°	Wrists and hands	Endocarditis	Present	Mitral systolic murmur, apex normal	15	„	Hst. pot. cit.	—
16 A. N.	M.	17	July	28th	1	16	Grandfather had rheumatism, maternal uncle gout, paternal great aunt insane	4	5	100·2°	Knees, shoulders	—	—	Faint apex, systolic murmur ? functional	8	„	Hst. pot. cit.	Sore throat at commencement.

TABLE C—*continued.*

No. and Name.	Sex.	Age.	Month admitted	Day of illness admitted	Previous attacks.	Age when first attacked.	Family history.	Days of fever in hospital.	Days of pain in hospital.	Highest temp.	Joints, &c., affected.	Heart.			No. of days in hospital	Result.	Treatment	Remarks.
												Affection.	In which attack developed.	State on discharge.				
17 G. J.	M.	6	July	3rd	0	6	Father has rheumatism	9	2	100·6°	Knees, left wrist, and fingers	Endocarditis	Present	Aortic and mitral systolic murmurs	10	Recovery	Hst. pot. cit.	Erythematous rash over arms lasting 2 days.
18 M. A. C.	F.	21	„	4th	1	5	2 paternal uncles have had acute rheumatism, maternal aunt died of phthisis	18	15	104°	Ankles, knees, left hip, left shoulder, wrist	Endocarditis	Previous and present	Double aortic and double mitral murmurs	109	„	Hst. sod. sal., hst. pot. cit.	Got very wet 14 days before. Albuminuria on admission. The symptoms during the greater part of her stay were purely cardiac.
19 T. R.	M.	11	„	3rd	0	11	—	11	1	104·6°	Knees, very slight	Endocarditis	Present	Aortic systolic murmur	21	„	Hst. pot. cit., hst. sod. sal.	—
20 W. M.	M.	15	„	6th	2	14	Father hadrheumatic gout and died insane, mother has epilepsy,	24	16	103°	Left ankle, both knees, fingers	Pericarditis, endocarditis	Previous and present	Mitral systolic murmur, apex ½in. outside nipple	50	„	Hst. pot. cit.	Wet through a week before. Last attack in December, 1889. (No. 47, Table C.)

							1 sister has caries of the spine, and 1 has had acute rheumatism										
21 A. R.	F.	28	„	4th	2	9	Father, mother, and a sister died of phthisis	14	13	103·4°	Both knees and ankles	Pericarditis	Present	Heart sounds normal	47	„	Hst. sod. sal.	Albuminuria at commencement.
22 R. B.	M.	28	Aug.	12th	0	28	1 sister has had rheumatism	31	29	103·4°	Ankles, knees, wrists, shoulders	Pericarditis, endocarditis	Previous and present	Mitral systolic murmur, apex 1½ in. outside nipple in 4th space	32	„	Hst. sod. sal., hst. pot. cit.	—
23 H. W.	F.	40	„	4th	1	29	—	6	21	103°	Left shoulder, both knees, left elbow, and wrist	Endocarditis	Previous	Double mitral murmur, apex in nipple line and 5th space	39	„	Hst. sod. sal.	—
24 R. U.	M.	29	„	6th	2	15	—	36	30	103·6°	Both hips, knees, ankles, great toes, wrists	Pericarditis, endocarditis	Previous and present	Double aortic and mitral systolic murmurs	61	„	„	A policeman. Tonsillitis a month before.

TABLE C—*continued.*

No. and Name.	Sex.	Age.	Month admitted	Day of illness admitted	Previous attacks.	Age when first attacked.	Family history.	Days of fever in hospital.	Days of pain in hospital.	Highest temp.	Joints, &c., affected.	Heart.			No. of days in hospital.	Result.	Treatment	Remarks.
												Affection.	In which attack developed.	State on discharge.				
25 A. G.	M.	25	Aug.	7th	0	25	Father died of phthisis	8	9	102·2°	Ankles, knees, hips	—	—	—	10	Recovery	Hst. sod. sal.	—
26 E. K.	F.	16	Sept.	8th	0	16	—	25	7	104°	Knees, ankles, wrists, hands	Pericarditis, endocarditis	Present	Mitral systolic murmur	49	„	Hst. sod. sal., hst. pot. iod. alk.	A very large pericardial effusion which persisted for some weeks, but caused very little distress.
27 M. A. R.	F.	53	„	21st	1	29	—	28	26	102·4°	Feet, hands, knees, elbows	Endocarditis	Previous?	Mitral systolic murmur	35	„	Hst. pot. cit.	—
28 A. H.	F.	28	„	7th	1	18	Mother had acute rheumatism, act. 7	10	8	104·4°	Hands, left shoulder, ankles, knees	Endocarditis	Previous	Double mitral murmur, apex in nipple line	23	„	Hst. sod. sal., pulv. ipecac. co.	Rather severe bronchitis.
29 C. F.	M.	15	„	5th	0	15	Mother and 1 sister have had rheumatism	8	10	102·2°	Hips, knees, ankles, left shoulder, wrist	Endocarditis	Present	Mitral systolic murmur	41	„	Hst. sod. sal.	—
30 M. McD.	F.	39	„	4th	1	12	Father has phthisis	49	55	104°	Hips, knees, ankles, shoulders, elbows, wrists, fingers	Endocarditis	„	Mitral systolic murmur	79	„	Hst. sod. sal., pulv. ipecac. co.	A stout woman. Very profuse sweating.

31 G. B.	M.	14	„	14th	0	14	—	13	7	101·4°	Knees, left ankle, very slight	Pericarditis, endocarditis	Previous and present	Mitral systolic murmur, apex in nipple line	14	„	Hst. pot. cit.	—
32 R. C.	F.	10	„	2nd	1	10	Father had acute rheumatism once, paternal uncle plumbism and gout, maternal grandfather died of phthisis	9	11	101·2°	No joint pains, numerous nodules on knuckles, elbows, clavicles, spine of scapula, and vertebral spines	„	„	Gradual heart failure. P.M. 11½ oz. blood-stained fluid in pericardium. Much thick spongy lymph. Adherent in parts, recent endocarditis of mitral, tricuspid and aortic valves, partial adhesion of left pleura	11	Death	Blisters, brandy	Chorea (no apparent cause) 2 months ago, followed by acute rheumatism. Never well since.
33 M. N.	M.	17	„	2nd	0	17	—	9	12	101·8°	Knees, ankles, wrists, elbows	—	—	Heart sounds normal	24	Recovery	Hst. sod. sal.	Got wet through 3 days before.

TABLE C—*continued.*

No. and Name.	Sex.	Age.	Month admitted	Day of illness admitted	Previous attacks.	Age when first attacked.	Family history.	Days of fever in hospital.	Days of pain in hospital.	Highest temp.	Joints, &c., affected.	Heart.			No. of days in hospital.	Result.	Treatment	Remarks.
												Affection.	In which attack developed.	State on discharge.				
34 A. C.	M.	11	Sept.	7th	0	11	Maternal grandmother died of phthisis, father has gout	3	5	102·4°	Left knee, ankles	? Faint apex systolic murmur	Present	Heart sounds normal	18	Recovery	Hst. pot. cit.	—
35 M. B.	F.	17	„	14th	1	11	—	18	14	105·4°	Knees, ankles, shoulders	Endocarditis, pulmonary functional bruit	„	Mitral systolic (organic) and pulmonary systolic (functional) murmurs	32	„	Hst. sod. sal.	Temp. rose rapidly on one occasion to 105·4° and hyperpyrexia seemed imminent, but it quickly fell again after large dose of sod. salicylat. Albuminuria at the same time.
36 A. H.	M.	22	„	3rd	0	22	—	4	4	102°	Right knee, ankle, wrist, and elbow	Pericarditis, endocarditis	„	Mitral and aortic systolic murmurs	23	„	„	—
37 H. S.	M.	42	Oct.	21st	0	42	—	14	12	102·4°	Fingers, knees, wrists	Endocarditis	„	Mitral systolic murmur	53	„	Hst. pot. iod., hst. sod. sal.	Simulated gout at commencement. Slightly maniacal at first.
38 W. M.	M.	16	„	2nd	3	14	See previous admission, No. 20	4	4	101·4°	Ankles, knees, hips, wrists	„	Previous	„	18	„	Hst. pot. cit.	—

39 H. W.	M.	12	„	4th	2	5	—	3	4	99·8°	Both knees and ankles, right shoulder	Pericarditis, endocarditis	?	„	19	„	Hst. sod. sal.	—
40 F. J.	M.	15	„	42nd	1	14	Father and paternal uncle have had acute rheumatism, 1 paternal uncle gout	4	4	101·4°	Wrists, knees	Endocarditis	Previous	Mitral systolic murmur, apex in nipple line	15	„	Hst. pot. cit.	—
41 E. J.	F.	7	„	In hospital	0	7	Mother has had acute rheumatism 3 times	13	2	104·2°	Shoulders very slightly	Pericarditis, endocarditis	Present	Long mitral systolic murmur, apex in nipple line ? adherent pericardium	48	„	—	Was in St. Thomas's Hospital 7 weeks from Aug. 1st with chorea. Was admitted into Westminster Hospital for heart affection only. Acute rheumatism came on while in hospital.
42 S. E.	F.	10	„	5th	0	10	Mother has rheumatism	9	9	103·8°	Knees, ankles, elbows, wrists	„	„	Persisting pericardial friction, mitral systolic murmur	45	„	Hst. pot. cit., pulv. ipec. co.	Double lobar pneumonia very bad at first, but rapid improvement. Illness probably aggravated by being sent to school by mother after onset of rheumatism, on the ground of shamming.
43 W. H.	M.	24	„	4th	1	22	—	8	14	101·8°	Left wrist, shoulders	Pericarditis, endocarditis (? ulcerative)	Present and previous ?	Double aortic and systolic murmurs, apex in 5th interspace 1 in. outside nipple	72	„	Hst. sod. sal., liq. strychninæ	Very ill for first 6 weeks with symptoms of acute aortic incompetence. Several very severe attacks of syncope. Then great improvement. Discharged in 1891.

TABLE C—*continued.*

No. and Name.	Sex.	Age.	Month admitted	Day of illness admitted	Previous attacks.	Age when first attacked.	Family history.	Days of fever in hospital.	Days of pain in hospital.	Highest temp.	Joints, &c., affected.	Heart.			No. of days in hospital.	Result.	Treatment	Remarks.
												Affection.	In which attack developed.	State on discharge.				
44 G. W.	F.	16	Oct.	21st	2	10	Father has rheumatism, 2 paternal uncles died of acute rheumatism	2	4	102·2°	Knees, ankles, fingers	Endocarditis	Previous	Mitral systolic murmur, apex in nipple line, 4th interspace	21	Recovery	Hst. sod. sal.	Previous admission. No. 39 (Table C), 1889.
45 G. W.	M.	15	„	5th	1	10	Father has gout	17	6	101·8°	Ankles, shoulders	Pericarditis, endocarditis	Previous and present	Double aortic and mitral systolic murmurs, apex in nipple line, 5th interspace	33	„	„	Lapsed into a state of almost insane irritability during convalescence.
46 H. S.	F.	12	„	21st	0	12	Father has spinal curvature, maternal aunt epilepsy	7	2	103·6°	Ankles, knees, hips, shoulders, very slightly	Pericarditis (slight) ? endocarditis	Present	Heart sounds normal	21	„	Pil. opii.	Erythema nodosum on legs.

47 H. L.	M.	10	„	4th	0	10	Father and maternal uncle died of phthisis	5	1	102·8°	Shoulders	Pericarditis, endocarditis	„	Mitral systolic murmur	20	„	—	—
48 A. H.	M.	24	Nov.	4th	1	18	Father had acute rheumatism several times, and died of phthisis, 1 brother died of phthisis, paternal uncle has gout	3	6	103·2°	Left elbow, wrist, both ankles and knees	—	—	Heart sounds normal	16	„	Hst. sod. sal.	—
49 C. S.	F.	31	„	5th	1	17	—	7	12	103°	Elbows, shoulders, wrists, knees, ankles	Pericarditis, endocarditis	Present	Mitral systolic murmur. apex in normal position	19	„	Hst. pot. cit., pulv. ipecac. co.	—
50 F. B.	F.	24	„	8th	1	24	Father and mother died of phthisis	40	35	104°	Knees, ankles, hips, shoulders, elbows, wrists	Pericarditis, endocarditis	„	Mitral systolic murmur, apex in 5th interspace and nipple line	67	„	Hst. sod. sal.	Most profuse crop of sudamina. Discharged in 1891.

TABLE C.—*continued.*

No. and Name.	Sex.	Age.	Month admitted	Day of illness admitted	Previous attacks.	Age when first attacked.	Family history.	Days of fever in hospital.	Days of pain in hospital.	Highest temp.	Joints, &c., affected.	Heart.			No. of days in hospital.	Result.	Treatment	Remarks.
												Affection.	In which attack developed.	State on discharge.				
51 W. J.	M.	17	Nov.	4th	1	16	Mother had acute rheumatism and died, as also her sister, of phthisis	4	7	103·6°	Ankles, knees, shoulders	Pericarditis? endocarditis	Present	1st sound prolonged	46	Recovery	Hst. pot. cit., hst. sod. sal.	Slight albuminuria.
52 C. C.	F.	36	„	11th	0	36	Mother died of phthisis	3	12	102·2°	Ankles, knees, knuckles	Faint apex systolic murmur ? functional	„	Heart sounds normal	24	„	Brandy, hst. sod. sal.	Would not take proper food before admission, and was therefore extremely prostrate; sordes on lips, etc., at first. Soon improved.
53 F. O.	M.	22	„	9th	0	22	Father had acute rheumatism and died of morbus cordis. He also had fibroid phthisis from inhalation of glass dust	12	7	102°	Kness, ankles, left elbow, wrists	Pericarditis, endocarditis	„	Mitral and (?) aortic systolic murmurs	34	„	Hst. sod. sal.	Got wet through 2 days before.

54 L. P.	F.	14	„	15th	0	14	Father has rheumatism	6	3	101°	Knees, ankles, elbows, hands, no nodules	Endocarditis	„	Very faint mitral systolic murmur	20	„	Hst. sod. sal., hst. ferri. cit.	Commenced with sore throat.
55 J. A.	M.	20	„	6th	2	15	1 sister has phthisis	11	5	102·4°	Right knee, both ankles	Pericarditis? endocarditis	Previous and present	Double aortic and mitral systolic murmurs	40	„	Hst. pot. cit., brandy	Began with sore throat. Developed right lobar pneumonia, sleepless, pulse intermittent. Good recovery. Discharged in 1891.
56 W. R.	M.	16	Dec.	5th	0	16	A brother died of phthisis æt 30	3	3	102°	Both ankles and knees	—	—	Heart sounds normal	25	„	Hst. pot. cit.	—
57 T. H.	M.	21	„	5th	1	9	—	10	7	103°	Both hands, wrists, ankles	Pericarditis, endocarditis	Present	Rough systolic murmur at apex. Beat ½ in. inside nipple	30	„	Hst. sod. sal., pulv. ipecac. co.	Discharged in 1891. Slight relapse of 5 days.
58 E. G.	F.	29	„	21st	1	28	Father and 1 brother had acute rheumatism, mother died of bronchitis, maternal grandmother had gout	20	16	102°	Ankles, knees, hands, shoulders	Slight pulmonary systolic murmur	„	Heart sounds normal	31	„	Hst. sod. sal., quinine, pulv. ipecac. co. fotus bellad.	Discharged in 1891.

TABLE C.—*continued.*

No. and Name.	Sex.	Age.	Month admitted	Day of illness admitted	Previous attacks.	Age when first attacked.	Family history.	Days of fever in hospital.	Days of pain in hospital.	Highest temp.	Joints, &c., affected.	Heart.			No. of days in hospital.	Result.	Treatment	Remarks.
												Affection.	In which attack developed.	State on discharge.				
59 C. Mc K.	F.	9	Dec.	11th	0	9	2 maternal aunts had acute rheumatism, paternal uncle and aunt died of fits, 1 brother has epilepsy	12	3	101·8°	Ankles, no nodules	Pericarditis, endocarditis	Present	Death from heart failure. P.M. Heart and pericardium weighed 16½ oz. Pericardium much thickened, surfaces covered with rough shaggy lymph and partly adherent, some fluid. A few vegetations on mitral valve. Double pleural effusion	19	Death	Brandy	Left off underclothing 2 days before the attack, during rain and snow, shivered afterwards. 2 relapses, with fresh pericarditis. —

Table D.—*Chorea.*

During the years 1881-2-3-4-5-6-7-8-9-1890 197 cases of chorea have been treated in the Hospital. A summary of these cases is prefixed to the analysis of the cases of the year 1890.

Inheritance.

Rheumatism	69
Neurosis	61
Phthisis	22
Gout	8

Sex.

	Males.	Females.
1881	6	14
1882	4	18
1883	3	14
1884	11	17
1885	7	12
1886	5	11
1887	12	12
1888	6	13
1889	3	10
1890	5	14
Total	62	135

Ages.

	Under 5.	5—10.	10—20.	20—30.	30—40.	40—50.	50—60.
1881	0	4	17	1	0	0	0
1882	0	4	13	2	1	0	0
1883	1	4	10	2	0	0	0
1884	0	8	17	2	0	0	1
1885	0	3	14	1	0	1	0
1886	0	6	8	2	0	0	0
1887	2	5	17	0	0	0	0
1888	0	5	14	0	0	0	0
1889	0	2	10	1	0	0	0
1890	0	4	13	2	0	0	0
Total	3	45	133	13	1	1	1

Rheumatism.—There was a history of acute rheumatism in 59 cases. The relation of the acute rheumatism to the chorea was as follows:—In 19 cases the rheumatism was suggested as the cause; in 14 cases the attack of rheumatism had developed during the course of the chorea; in the remaining 26 cases

there was a distinct interval of good health between the rheumatism and the chorea, viz., 6 years (2), 5 years, 4 years, 3 years (2), 2 years, 1 year (3), 7 months, 6 months (2), 5 months, 4 months, 3 months (3), 2 months (2), 6 weeks (2), 1 month (3). 14 days.

Causes.—In 119 cases of the whole the causes assigned by the friends were fright, sorrow, or overwork at school.

Heart.—In 37 cases a mitral murmur constant in force and rhythm was audible; of these 37 cases 22 had suffered acute rheumatism previously. In 84 cases a murmur was heard over the apex occasionally only, the murmur varying in time and intensity, and having quite disappeared at the discharge of the patient.

TABLE D—*continued.*

No. and Name.	Family history.	Previous illness with special reference to rheumatism.	Previous chorea.	Sex.	Age.	Heart condition.		Part of body affected.	Cause.	Result.	Number of Days in Hospital.	Remarks.
						In Hospital.	On Discharge.					
1 E. B.	Father æt 70, suffers from rheumatism, 5 brothers and 6 sisters died of phthisis	Acute rheumatism in hands, feet and knees, 6 weeks before admission, herpes zoster after she went back to work	0	F.	15	Mitral systolic murmur heard at angle of scapula, heart's action irregular, slight pericarditis 6 weeks after admission	Mitral systolic murmur	Both arms, face, legs slightly. Very bad at first, bed padded, &c. Then rapid improvement, but being frightened by a Tierra del Fuegian woman in the ward, she became much worse again. Movements most in the feet, 2nd attack, this attack was as bad as the 1st, and recovery was slow. During this she became very emotional	None known	Relief	68	Catamenia commenced 9 months ago, ceased after 2 months. Pulv. Ipecac. co.

2 M. W.	Mother died of phthisis	Had acute rheumatism 2 months before admission, pains ceased 1 month ago	0	M.	12	Systolic murmur at apex, heart's action irregular	Blowing systolic murmur at apex, beat in nipple line	Right arm, leg and side of face. The day after admission was much frightened by a death in the next bed, movements became much worse, and general; speech much affected	Was caned twice a day at school for 3 weeks before the attack because he did not know his work. After onset upset a lamp and became much worse	Relief	27	Very emotional, but intelligent.
3 F. D.	Mother has rheumatism	In Westminster Hospital 2 years ago with tuberculosis	0	M.	16	Faint systolic murmur at apex, heart's action irregular	Heart sounds clear	Right arm, both legs, face and tongue	None known	Relief	101	Dulness and bronchial breathing at right apex, 6 weeks after admission became very violent (maniacal), and gradually improved.
4 J.E. D.	Maternal uncle died of acute rheumatism, a sister is in an asylum, a brother has gout	Had had and still complained of pains in joints, no swelling	0	F	12	Heart's action slightly uneven	Heart's action regular and sounds clear	General, more marked on left side speech considerably affected	Excited over some acting at school a short time before the movements began, and a few days after dropped the baby	Relief	37	Catamenia not commenced.

TABLE D—*continued.*

No. and Name.	Family history.	Previous illness, with special reference to rheumatism.	Previous chorea.	Sex.	Age.	Heart condition.		Part of body affected.	Cause.	Result.	Number of Days in Hospital.	Remarks.
						In Hospital.	On Discharge.					
5 C. L.	Mother has had acute rheumatism	Has had measles; scarlet fever during last attack of chorea in hospital	3	F.	12	Heart's action irregular	Heart's action regular and sounds clear	General, more marked in right arm, face and tongue	She saw a man injured at football and thought he was killed the day before the movements came on	Relief	11	Prev. adm. No. 5, table D, 89, brought on by fright from Guy Fawkes.
6 H. M.	—	Some stiffness of joints a month previous	0	M.	13	Heart's action slightly uneven, soft systolic murmur at all orifices	Heart sounds clear	Right arm, leg (less) face and tongue slightly	None known	Relief	8	Slight case.
7 C. M.	Mother has rheumatism, father has gout, 2 sisters died of phthisis	Gout for 16 weeks, 6 months ago, measles twice	3	F.	22	Soft systolic murmur at apex	Systolic murmur at apex	Left arm, and leg, face and tongue slightly	Worrying about nursing her brothers and sisters	Relief	3	First attack æt 7 after a blow from her father; married five years, no children, rapid improvement.
8 M. P.	Father has rheumatism, maternal grandfather epilepsy.	—	0	F.	13	Systolic murmur all over præcordia, heart's action uneven	Heart sounds clear, action uneven	General, speech much affected	Frightened 4 months previously by seeing a rat. Chorea came on 6 weeks previous to admission	Relief	44	Very rapid improvement.

9 C. L.	No history obtained	Influenza 1 month, and pains and swelling in ankles 14 days previous	0	F.	15	Systolic murmur at all orifices, especially mitral	Systolic murmur at apex, beat in normal position	General, speech much affected, considerable paresis of legs latterly	Frightened by a girl fainting 3 days previous	Relief	68	Very slow improvement, Liq. Arsenicalis ♏ iij. for some time.
10 E. N.	—	Measles	0	F.	8	Systolic murmur at all orifices, action uneven	Heart sounds clear	General	Excitement about examination (possibly)	Relief	65	Very emotional and then extremely obstinate, refusing to speak for weeks. Relapsed again for a few days at the end after some excitement.
11 E. S.	Paternal grandfather and grandmother died of phthisis	Measles, whooping cough	0	F.	12	Short systolic murmur at apex, action slightly irregular	Remains the same	Left arm and leg, face; right side became affected later	Had to work hard, no special cause	Relief	43	Catamenia not commenced.
12 A. B.	—	,,	3	F.	12	Systolic murmur at ensiform cartilage, action irregular	Heart sounds clear	General, more on left side	None known	Relief	12	Slight case, first attack came on after being frightened by a beating.
13 K. E.	—	Measles twice.	0	F.	6	Faint systolic murmur at apex, action uneven	Heart sounds clear, action uneven	Face, tongue and arms, right leg afterwards became paretic	Teacher hit her a week previously	Relief	17	Slight case.
14 E. W.	Paternal aunt has rheumatism, maternal aunt has fits	Measles twice, whooping cough	1	F.	16	Occasional systolic murmur at apex, action regular	Faint systolic murmur at apex	General, more on right side, and legs very slightly affected	None known	Relief	65	Had tonsillitis during attack, after which movements became worse, followed by a slight attack of acute rheumatism. First attack caused by over study.

TABLE D—*continued.*

No. and Name.	Family history.	Previous illness, with special reference to rheumatism.	Previous chorea.	Sex.	Age.	Heart condition.		Part of body affected.	Cause.	Result.	Number of Days in Hospital.	Remarks.
						In Hospital.	On Discharge.					
15 J. F.	Father has epileptic fits, paternal grandfather committed suicide while melancholic	Fell off a van 3 months previously, insensible some time	0	M.	14	Heart's action slightly irregular, sounds clear	Heart sounds clear	General and very severe, followed by considerable paresis, then rapid recovery towards the end	Saw a great friend run over and killed by an engine a few days previous	Recovery	64	Very emotional; 4 weeks after admission, when considerable paresis of legs and arms was present, electrical reactions of both nerves and muscles were normal, though faradic contractions were not very brisk.
16 J. D.	Father suffers from gout, maternal first cousin died of phthisis	Pain and swelling of left hand just before attack	0	F.	21	Loud systolic murmur at apex, heard also at base, action regular	Loud systolic murmur at apex, apex normal position	Left arm, leg, face, tongue, speech much affected, right hand slightly	Was frightened 5 weeks before admission by her brother-in-law striking her sister, movements came on a fortnight after this	Relief	57	Hst. Potass. Bromid.
17 E. A.	Father died of phthisis	Bronchitis several times, sore throat a week before admission	0	F.	9	Heart sounds clear	Heart sounds clear	Movements general, speech good	When she returned to school after her sore throat she could not write owing to	Slight relief	15	Very emotional, often crying for 3 or 4 hours together. So noisy at last that she had to be sent out.

									movements of her hand, she was therefore struck by the teacher, movements then became worse			
18 R. N.	A brother has epilepsy	Acute rheumatism just before 1st attack of chorea æt 6, and present chorea came on during acute rheumatism	1	M.	15	Systolic murmur at apex, apex beat ½ in. inside nipple in 5th space	Mitral systolic murmur	General, especially arms, face and tongue, speech much affected, severe case at first, then rapid improvement	Came on during an attack of acute rheumatism	Recovery	28	Pericarditis during chorea, before admission.
19 A. R.	—	In St. Margarets ward with chorea 12 months ago, pains in joints off and on since	1	F.	9	Loud rough apex systolic murmur and systolic thrill, apex ½ in. outside nipple in 4th interspace ? pericarditis	Occasional rough systolic murmur at apex, apex in 5th interspace inside nipple	Movements general, face and speech much affected	Movements came on as she was recovering from the rheumatic pains	,,	22	Nodule on knuckle of each hand. Fright caused first attack. Previous admission, No. 13, Table D, 89.

TABLE E.—*List of Deaths in the Medical Wards.*

	Males.		Females.
GENERAL DISEASES:			
Erysipelas	1	...	0
Enteric fever	0	...	1
Diphtheria	2	...	6
Morbilli	1	...	1
Pertussis	0	...	1
Syphilis	0	...	1
Acute rheumatism	0	...	2
Tuberculosis	7	...	8
Hodgkin's disease	1	...	0
DISEASES OF THE NERVOUS SYSTEM:			
Cerebral hæmorrhage	2	...	0
„ abscess	1	...	0
„ tumour	1	...	0
„ softening after thrombosis of sylvian artery	1	...	0
Thrombosis of lateral sinus	0	...	1
Meningitis	4	...	2
Hydrocephalus	0	...	1
Laryngismus Stridulus	1	...	0
Delirium tremens	1	...	0
DISEASES OF THE CIRCULATORY SYSTEM:			
Ulcerative endocarditis	1	...	0
Mitral disease	5	...	6
Aortic disease	1	...	2
Pericarditis	0	...	1
Thoracic aneurysm	4	...	1
Abdominal aneurysm	1	...	0
DISEASES OF THE RESPIRATORY SYSTEM:			
Acute bronchitis	1	...	1
Chronic „	1	...	0
Lobar pneumonia	14	...	2
Lobular „	6	...	4
Phthisis	16	...	7
Emphysema	4	...	0
Gangrene of the lung	0	...	1
Pleural effusion	1	...	0
Empyema	1	...	2
DISEASES OF THE DIGESTIVE SYSTEM:			
Tonsillitis	1	...	0
Rupture of œsophagus	1	...	0
Scirrhus of „	0	...	1
Gastric ulcer	0	...	1

	Males.		Females.
DISEASES OF THE DIGESTIVE SYSTEM—*continued.*			
Carcinoma of stomach	2	...	1
Duodenal ulcer	1	...	0
Intestinal stricture from band	0	...	1
Perforation of vermiform appendix	2	...	0
Diarrhœa	2	...	2
Acute hepatitis	1	...	0
Cirrhosis of liver	1	...	1
Malignant disease of liver	0	...	1
Impacted gall stone	0	...	1
Chronic peritonitis	1	...	1
Malignant disease of peritoneum	1	...	0
Marasmus	3	...	3
Sarcoma of pelvis	1	...	0
DISEASES OF THE URINARY SYSTEM:			
Chronic nephritis, large white kidney	2	...	1
„ „ contracting granular kidney	2	...	1
Suppurative nephritis	1	...	0
Total	102	...	67

SURGICAL REGISTRAR'S REPORT FOR THE YEAR 1890.

By W. W. POWELL.

The total number of cases under treatment in the surgical wards during the past year was 1288 viz.:

	Males.	Females.	Total.
Remaining in hospital December 31st, 1890 ...	37	33	70
Admitted during the year 1890			1218
			1288
Of these there were:			
Discharged, cured, or relieved	681	425	1106
„ unrelieved	25	16	41
Transferred to medical wards	5	4	9
Died	32	25	57
In hospital December 31st, 1890	51	24	75
			1288

The mortality during the year was 4·42 per cent.

TABLE I.—*Showing the total number of Cases of each Disease or Injury which were Discharged or Died during the year* 1890.

DISEASES.	Total.	Relieved.		Not Relieved.		Died.		In hosp. 31st Dec., 1890.	
		M.	F.	M.	F.	M.	F.	M.	F.
Measles	3	...	3	...	...	...	...	...	...
Purpura	1	...	1	...	...	...	...	...	...
Pneumonia	1	...	...	...	...	...	...	...	1
Erysipelas (cutaneous):									
i. Admitted	1	1	...	...	...	...	...	...	...
ii. Arising in hospital	1	1	...	...	...	...	...	...	...
Pyæmia (1. Wound of hand; 1. After amptn. of breast)	2	...	1	...	...	...	1	...	...
Rickets	1	...	1	...	...	...	...	...	...
Scabies	1	1	...	...	...	...	...	...	...
Rheumatism (subacute)	2	1	1	...	...	...	...	...	...
Gout	2	2	...	...	...	...	...	...	...
Syphilis (unclassified):									
Ulcers	6	2	3	...	1	...	...	...	...
Gummata	4	3	1	...	...	...	...	...	...
Pustular syphilide	1	1	...	...	...	...	...	...	...
MALFORMATIONS, and acquired Deformities:									
Cleft palate	4	1	2	1	...	...	...	...	...
Genu varum	3	...	2	...	...	...	...	1	...
Pes cavus	1	1	...	...	...	...	...	...	...
„ planus	5	3	1	1	...	...	...	...	...
Talipes varus	5	1	4	...	...	...	...	...	...
„ equino-varus and equinus	5	3	2	...	...	...	...	...	...
„ valgus	2	...	2	...	...	...	...	...	...
„ equino-valgus	1	...	1	...	...	...	...	...	...
Hammer-toe	4	3	...	...	...	...	...	1	...
Webbed-fingers	1	1	...	...	...	...	...	...	...
Stricture of rectum (congenital)	1	1	...	...	...	...	...	...	...
Angular ulna after fracture	1	...	1	...	...	...	...	...	...
Contraction after burn	1	1	...	...	...	...	...	...	...
DISEASES OF THE NERVOUS SYSTEM:									
Sequela of contusion of brain	1	...	...	1	...	...	...	...	...
Apoplexy	1	...	...	...	...	1	...	...	...
Cerebral abscess	1	...	...	...	...	1	...	...	...
„ softening	1	...	...	...	...	1	...	...	...
Delirium tremens	2	...	...	...	...	2	...	...	...
Hysteria	2	...	2	...	...	...	...	...	...
Sciatica	4	3	1	...	...	...	...	...	...
Clonic torticollis	1	...	...	1	...	...	...	...	...
Sunstroke	1	1	...	...	...	...	...	...	...
Neuroma	1	1	...	...	...	...	...	...	...

TABLE I.—*continued.*

DISEASES.	Total.	Relieved.		Not Relieved.		Died.		In hosp. 31st Dec., 1890.	
		M.	F.	M.	F.	M.	F.	M.	F.
DISEASES OF THE EAR:									
Otitis media purulenta	5	4	1	...	...	...	...	...	...
Mastoid abscess	5	3	2	...	...	...	...	...	...
DISEASES OF THE EYE and its Appendages:									
Blepharitis, specific	1	...	1	...	...	...	...	...	...
Gumma of eyelid	1	1	...	...	...	...	...	...	...
Trichiasis	1	...	1	...	...	...	...	...	...
Ectropion	1	...	1	...	...	...	...	...	...
Entropion	1	1	...	...	...	...	...	...	...
Suppuration of meibomian follicles	1	1	...	...	...	...	...	...	...
Dacryocystitis	1	1	...	...	...	...	...	...	...
Strabismus	2	1	1	...	...	...	...	...	...
Sympathetic ophthalmia	1	1	...	...	...	...	...	...	...
Conjunctivitis	1	...	1	...	...	...	...	...	...
Keratitis	3	1	1	...	...	...	...	1	...
Corneal ulcer	3	2	1	...	...	...	...	...	...
Sloughing cornea	1	...	1	...	...	...	...	...	...
Nebulæ	1	...	1	...	...	...	...	...	...
Iritis	2	2	...	...	...	...	...	...	...
Kerato-iritis	4	4	...	...	...	...	...	...	...
Anterior synechiæ	5	3	2	...	...	...	...	...	...
Cataract	11	3	7	...	...	...	...	1	...
Glaucoma	1	...	1	...	...	...	...	...	...
Albuminuric retinitis	1	1	...	...	...	...	...	...	...
Optic neuritis	2	...	...	2	...	...	...	...	...
Traumatic panophthalmitis	3	2	1	...	...	...	...	...	...
Disorganization of globe	6	4	2	...	...	...	...	...	...
DISEASES OF THE NOSE, and Naso-pharynx:									
Ozena	1	...	...	...	1	...	...	...	...
Abscess in roof	1	1	...	...	...	...	...	...	...
Mucous polypus	1	...	1	...	...	...	...	...	...
Hypertrophied mucous membrane	1	...	1	...	...	...	...	...	...
Adenoid vegetations	14	8	6	...	...	...	...	...	...
DISEASES OF THE CIRCULATORY SYSTEM:									
Thrombosis and phlebitis	8	4	3	...	...	...	...	...	1
Varicose veins	7	3	3	...	1	...	...	...	...
Hæmorrhage from varicose vein	1	...	1	...	...	...	...	...	...
Hæmatoma	1	...	1	...	...	...	...	...	...
Aneurysm, femoral	1	1	...	...	...	...	...	...	...
,, subclavian	1	...	...	...	...	1	...	...	...
Epistaxis	2	1	1	...	...	...	...	...	...
Hæmophilia	1	1	...	...	...	...	...	...	...

TABLE I.—*continued.*

DISEASES.	Total.	Relieved.		Not Relieved.		Died.		In hosp. 31st Dec., 1890.	
		M.	F.	M.	F.	M.	F.	M.	F.
DISEASES OF THE RESPIRATORY SYSTEM :									
Acute laryngitis	1	1	...	...	...	...	...	...	...
Gumma of larynx	1	1	...	...	...	...	...	...	...
Dyspnœa from pressure of cancerous glands on trachea	1	1	...	...	...	...	...	...	...
Empyema	6	2	...	1	...	1	1	...	1
Emphysema of chest wall (traumatic)	2	2	...	...	...	...	...	...	...
DISEASES OF THE LYMPHATIC SYSTEM :									
Glands :									
i. Strumous enlargement	6	3	2	...	...	...	...	1	...
ii. Inflammation	23	10	10	1	...	...	...	1	1
iii. Suppuration	11	4	6	...	...	...	...	...	1
Lymphangitis	7	4	3	...	...	...	...	...	...
DISEASES OF THE THYROID GLAND :									
Bronchocele (exophthalmos)	1	...	...	...	...	...	1	...	...
Abscess	2	2	...	...	...	...	...	...	...
DISEASES OE THE DIGESTIVE SYSTEM :									
Lips :									
Epithelioma	4	3	...	...	...	1	...	...	...
Jaws :									
Epithelioma, lower jaw	1	...	...	...	...	1	...	...	...
Tongue :									
Tubercular ulcer	1	1	...	...	...	...	...	...	...
Chronic ulcer	1	...	1	...	...	...	...	...	...
Epithelioma	4	2	1	1	...	...	...	...	...
„ recurrent	1	...	...	...	...	...	...	1	...
Ulcerative stomatitis	1	...	1	...	...	...	...	...	...
Tonsils :									
Tonsillitis	2	1	1	...	...	...	...	...	...
Chronic enlargement	2	2	...	...	...	...	...	...	...
Hæmorrhage after excision	1	1	...	...	...	...	...	...	...
Pharynx :									
Retropharyngeal gumma	1	...	1	...	...	...	...	...	...
Œsophagus :									
Malignant stricture	5	...	1	...	1	1	2	...	...
Peritoneum :									
Peritonitis	3	...	...	...	1	2	...	...	...
Ascites	1	...	...	...	1	...	...	...	...

TABLE I.—*continued.*

DISEASES.	Total.	Relieved.		Not Relieved.		Died.		In hosp. 31st Dec., 1890.	
		M.	F.	M.	F.	M.	F.	M.	F.
DISEASES OF THE DIGESTIVE SYSTEM—*continued.*									
Intestines:									
Hernia (not strangulated):									
Reducible	10	8	2	...	...	...	...	...	...
Irreducible	4	2	2	...	...	...	...	...	...
Hernia (strangulated):									
Inguinal	5	4	...	...	...	...	1	...	...
Femoral	5	...	...	...	...	...	5	...	...
Umbilical	2	...	2	...	...	...	...	...	...
Acute internal strangulation by band of adhesion	1	...	...	...	...	...	1	...	...
Carcinoma of colon	1	...	...	...	...	...	1	...	...
„ sigmoid flexure	2	...	...	...	...	...	2	...	...
Prolapse through artificial anus	1	...	...	1	...	...	...	...	...
Fæcal fistula	2	...	...	1	...	1	...	...	...
Rectum and Anus:									
Columnar epithelioma	3	1	1	...	...	1	...	...	...
Stricture (? cause)	1	1	...	...	...	...	...	...	...
Condylomata	2	1	1	...	...	...	...	...	...
Myxomatous polypus	1	1	...	...	...	...	...	...	...
Fistula	15	9	6	...	...	...	...	...	...
Hæmorrhoids	16	7	7	1	...	...	...	...	1
Fissure	2	2	...	...	...	...	...	...	...
Prolapsus	2	1	1	...	...	...	...	...	...
Lacerated perineum	1	...	1	...	...	...	...	...	...
DISEASES OF THE URINARY SYSTEM:									
Kidney:									
Calculus	2	1	1	...	...	...	...	...	...
„ suppurating	1	...	1	...	...	...	...	...	...
Bladder:									
Epithelioma	2	...	...	...	1	1	...	...	...
Villous growth	1	...	1	...	...	...	...	...	...
Cystitis	3	1	...	...	1	1	...	...	...
Calculus	4	3	...	1	...	...	...	...	...
Incontinence of urine	2	1	1	...	...	...	...	...	...
Prostate:									
Hypertrophy, with retention of urine	3	...	...	1	...	2	...	...	...
Urethra:									
Caruncle	2	...	2	...	...	...	...	...	...
Traumatic stricture	2	2	...	...	...	...	...	...	...
Perineal fistula (old lithotomy wound)	1	...	...	1	...	...	...	...	...

TABLE I.—*continued.*

DISEASES.	Total.	Relieved.		Not Relieved.		Died.		In hosp. 31st Dec., 1890.	
		M.	F.	M.	F.	M.	F.	M.	F.
DISEASES OF THE URINARY SYSTEM—*continued.*									
Gonorrhœa and its Complications:									
Gonorrhœa and balanitis...	3	3	...	...	...	...	...	...	...
Bubo	23	18	3	...	...	...	...	2	...
Stricture of urethra	15	13	...	1	...	...	...	1	...
Gonorrhœal warts.....	3	1	2	...	...	...	...	...	...
Perineal abscess	1	1	...	...	...	...	...	...	...
„ fistula	4	4	...	...	...	...	...	...	...
Retention of urine	4	4	...	...	...	...	...	...	...
Extravasation of urine.....	3	...	...	...	...	3	...	...	...
Synovitis.....	1	1	...	...	...	...	...	...	...
Vaginitis.....	1	...	1	...	...	...	...	...	...
Epididymitis	4	4	...	...	...	...	...	...	...
DISEASES OF THE MALE GENERATIVE ORGANS:									
Penis:									
Chancre	2	2	...	...	...	...	...	...	...
Chancroid	3	3	...	...	...	...	...	...	...
Phimosis (congenital) ...	10	10	...	...	...	...	...	...	...
„ (acquired)	14	14	...	...	...	...	...	...	...
Paraphimosis	5	5	...	...	...	...	...	...	...
Inflammation after circumcision	1	1	...	...	...	...	...	...	...
Tunica vaginalis:									
Hydrocele	5	4	...	...	...	...	...	1	...
Old hæmatocele.....	1	1	...	...	...	...	...	...	...
Cord:									
Varicocele	7	7	...	...	...	...	...	...	...
Testis:									
Undescended	1	1	...	...	...	...	...	...	...
Tubercular	4	2	...	1	...	...	...	1	...
Syphilitic	2	2	...	...	...	...	...	...	...
Inflammation.....	3	3	...	...	...	...	...	...	...
DISEASES OF THE FEMALE GENERATIVE ORGANS:									
Vulva:									
Papilloma	1	...	1	...	...	...	...	...	...
Abscess	2	...	1	...	...	...	...	...	1
Fibroma	1	...	1	...	...	...	...	...	...
Vagina:									
Vaginitis.....	1	...	1	...	...	...	...	...	...
Vesico-vaginal fistula ...	1	...	1	...	...	...	...	...	...
Uterus:									
Epithelioma cervix	1	...	...	...	1	...	...	...	...
Ovary:									
Cyst (*vide* report for Arden Ward)	1	...	...	...	...	...	1	...	...

TABLE I.—*continued.*

DISEASES.	Total.	Relieved.		Not Relieved.		Died.		In hosp. 31st Dec., 1890.	
		M.	F.	M.	F.	M.	F.	M.	F.
DISEASES OF THE FEMALE GENERATIVE ORGANS—*continued.*									
Breast:									
Acute abscess	6	...	6	...	...	...	...	...	...
Lobular induration	1	...	1	...	...	...	...	...	...
Cyst	1	...	1	...	...	...	...	...	...
Adeno-fibroma	5	...	5	...	...	...	...	...	...
Scirrhus, primary	9	1	6	...	...	...	2	...	...
„ recurrent	5	...	5	...	...	...	...	...	...
DISEASES OF JOINTS:									
Acute synovitis:									
Of knee	14	8	6	...	...	...	...	...	...
Chronic synovitis:									
Of knee	14	6	6	...	...	...	...	2	...
Of shoulder	1	1	...	...	...	...	...	...	...
Acute arthritis:									
Of knee	4	1	2	...	...	...	...	1	...
„ (Charcot's)	1	1	...	...	...	...	...	...	...
Tubercular arthritis:									
Of elbow	1	1	...	...	...	...	...	...	...
Of wrist	1	1	...	...	...	...	...	...	...
Of hip	32	14	9	...	1	1	1	3	3
Of knee	13	4	7	...	1	...	...	1	...
Of ankle	2	2	...	...	...	...	...	...	...
Chronic rheumatic arthritis	2	...	2	...	...	...	...	...	...
Fibrous ankylosis and adhesions:									
Of shoulder	3	1	2	...	...	...	...	...	...
Of elbow	3	2	...	1	...	...	...	...	...
Of hip	2	1	1	...	...	...	...	...	...
Of knee	1	1	...	...	...	...	...	...	...
Osseous ankylosis:									
Of elbow (old fracture)	3	...	...	1	1	...	...	...	1
"Flail" elbow (after excision)	1	1	...	...	...	...	...	...	...
„ knee „	1	...	1	...	...	...	...	...	...
Stiff knee (? hysteria)	1	...	1	...	...	...	...	...	...
DISEASES OF BONES:									
Acute ostitis and periostitis:									
Of radius	1	...	1	...	...	...	...	...	...
Of tibia	4	2	2	...	...	...	...	...	...
Chronic ostitis and periostitis:									
Of rib	1	1	...	...	...	...	...	...	...
Of tibia	1	1	...	...	...	...	...	...	...
Of metatarsus	2	1	...	...	...	...	...	1	...
Necrosis:									
Of nasal bones	1	...	1	...	...	...	...	...	...
Of upper jaw	3	1	2	...	...	...	...	...	...
Of lower jaw	7	2	4	1	...	...	...	...	...

TABLE I.—*continued.*

DISEASES.	Total.	Relieved.		Not Relieved.		Died.		In hosp. 31st Dec., 1890.	
		M.	F.	M.	F.	M.	F.	M.	F.
DISEASES OF BONES—*continued.*									
Necrosis—*continued.*									
Of sternum	1	...	...	...	...	...	...	1	...
Of rib	1	...	...	1	...	...	...	...	...
Of olecranon process	1	1	...	...	...	...	...	...	...
Of metacarpal bone	1	1	...	...	...	...	...	...	...
Of phalanges	5	5	...	...	...	...	...	...	...
Of ilium	1	...	1	...	...	...	...	...	...
Of femur	2	1	1	...	...	...	...	...	...
Of tibia	9	6	3	...	...	...	...	...	...
Of tarsus	1	...	...	...	...	...	...	1	...
Tubercular caries :									
Of spine	18	10	7	1	...	...	...	...	...
Of os calcis	3	...	3	...	...	...	...	...	...
Of tarsus	9	2	6	...	...	...	...	1	...
Of metatarsus	1	...	1	...	...	...	...	...	...
Tubercular dactylitis	3	3	...	...	...	...	...	...	...
Acute osteomyelitis of femur	1	1	...	...	...	...	...	...	...
Sarcoma of ilium	2	...	...	...	2	...	...	...	...
DISEASES OF MUSCLES, FASCIÆ, AND TENDON SHEATHS :									
Lateral curvature of spine	13	3	9	1	...	...	...	...	...
Spinal debility	1	...	1	...	...	...	...	...	...
Dupuytren's contraction of hand	2	2	...	...	...	...	...	...	...
Ganglion :									
Of wrist	4	2	2	...	...	...	...	...	...
Of finger	1	...	1	...	...	...	...	...	...
Of foot	1	...	1	...	...	...	...	...	...
DISEASES OF BURSÆ :									
Acute bursitis :									
Of olecranon bursa	4	4	...	...	...	...	...	...	...
Of prepatellar bursa	12	3	9	...	...	...	...	...	...
Of bursa under lig. patellæ	1	1	...	...	...	...	...	...	...
Chronic bursitis :									
Of prepatellar bursa	1	...	1	...	...	...	...	...	...
Suppuration of bursa under lig. patellæ	1	1	...	...	...	...	...	...	...
Enlargement of semimembranosus bursa	2	...	1	...	...	...	...	...	1
DISEASES OF THE SKIN AND SUBCUTANEOUS TISSUES :									
Psoriasis	1	1	...	...	...	...	...	...	...
Onychia	2	1	1	...	...	...	...	...	...
Eczema	1	...	1	...	...	...	...	...	...

TABLE I.—*continued.*

DISEASES.	Total.	Relieved.		Not Relieved.		Died.		In hosp. 31st Dec., 1890.	
		M.	F.	M.	F.	M.	F.	M.	F.
DISEASES OF THE SKIN AND SUBCUTANEOUS TISSUES—*continued.*									
Lupus	3	1	1	...	...	...	...	...	1
Carbuncle	4	3	1	...	...	...	...	...	...
Ingrowing toe-nail	3	2	1	...	...	...	...	...	...
Rodent ulcer	2	...	1	...	...	...	...	1	...
Erythema nodosum	2	1	1	...	...	...	...	...	...
Painful cicatrix	1	...	1	...	...	...	...	...	...
Papilloma of foot	1	1	...	...	...	...	...	...	...
Senile gangrene of foot	1	...	1	...	...	...	...	...	...
Nævus	8	5	2	...	...	...	...	1	...
Keloid	1	...	1	...	...	...	...	...	...
Epithelioma of nose	1	...	...	1	...	...	...	...	...
Lipoma of back	3	2	1	...	...	...	...	...	...
Sinus:									
In face	1	...	...	...	1	...	...	...	...
In loin (old nephro-lithotomy wound)	2	...	2	...	...	...	...	...	...
In knee	1	1	...	...	...	...	...	...	...
In leg	1	...	1	...	...	...	...	...	...
In foot	1	1	...	...	...	...	...	...	...
In great toe	1	...	1	...	...	...	...	...	...
In amputation stump	3	1	1	...	...	1	...	...	...
Cellulitis:									
Of face	2	1	1	...	...	...	...	...	...
Of scalp	2	2	...	...	...	...	...	...	...
Of neck	1	...	1	...	...	...	...	...	...
Of arm	6	3	2	...	...	...	...	1	...
Of hand	10	5	4	...	...	...	...	...	1
Of leg	3	...	1	...	...	...	...	2	...
Whitlow	22	10	11	...	...	...	...	1	...
Acute abscess:									
Of scalp	6	4	2	...	...	...	...	...	...
Of neck	26	11	13	...	...	1	1	...	...
Of face	7	4	1	...	...	...	...	1	1
Of chest wall	1	1	...	...	...	...	...	...	...
Of axilla	9	5	4	...	...	...	...	...	...
Of ischio-rectal fossa	13	9	3	...	1	...	...	...	...
Of hand	2	2	...	...	...	...	...	...	...
Of arm	2	1	1	...	...	...	...	...	...
Of abdominal wall	2	1	1	...	...	...	...	...	...
Of thigh (femoral glands)	1	1	...	...	...	...	...	...	...
Of popliteal space	1	1	...	...	...	...	...	...	...
Of leg	2	...	2	...	...	...	...	...	...
Of foot	8	5	2	...	...	...	...	1	...
Around shoulder joint	1	...	1	...	...	...	...	...	...

TABLE I.—*continued.*

DISEASES.	Total.	Relieved.		Not Relieved.		Died.		In hosp. 31st Dec., 1890.	
		M.	F.	M.	F.	M.	F.	M.	F.
DISEASES OF THE SKIN AND SUBCUTANEOUS TISSUES—*continued.*									
Around knee joint	3	...	1	...	...	...	...	1	1
Mastoid	1	1	...	...	...	...	...	...	...
Alveolar	1	1	...	...	...	...	...	...	...
Chronic abscess:									
Of neck	20	7	10	...	...	...	...	2	1
Of face	3	1	2	...	...	...	...	...	...
Of chest wall	2	2	...	...	...	...	...	...	...
Of back	3	1	2	...	...	...	...	...	...
Of arm	3	2	1	...	...	...	...	...	...
Of buttock	2	...	2	...	...	...	...	...	...
Of foot	1	1	...	...	...	...	...	...	...
Of leg	1	1	...	...	...	...	...	...	...
Mastoid	1	...	1	...	...	...	...	...	...
Of testicle (old injury)	1	1	...	...	...	...	...	...	...
Multiple tubercular abscesses	1	...	...	...	...	...	1	...	...
Simple ulcer:									
Of leg	3	1	2	...	...	...	...	...	...
Sloughing ulcer:									
Of arm (after vaccination)	1	1	...	...	...	...	...	...	...
Of leg	1	1	...	...	...	...	...	...	...
Varicose ulcer:									
Of leg	5	2	3	...	...	...	...	...	...
Traumatic ulcer:									
Of arm	1	...	1	...	...	...	...	...	...
Of leg	2	1	1	...	...	...	...	...	...
Syphilitic ulcer:									
Of leg	6	2	3	...	...	...	...	1	...
Multiple tubercular ulcers	1	...	...	...	...	...	...	...	1
Sebaceous cyst:									
Of scalp	2	1	1	...	...	...	...	...	...
Of forehead	1	1	...	...	...	...	...	...	...
Of neck	1	1	...	...	...	...	...	...	...
Serous cyst:									
Of face	1	...	1	...	...	...	...	...	...
Of neck	1	1	...	...	...	...	...	...	...
Dermoid cyst:									
At outer angle of orbit	1	1	...	...	...	...	...	...	...
Under chin	1	...	1	...	...	...	...	...	...
Cengenital cyst of buttock	1	1	...	...	...	...	...	...	...

TABLE I.—*continued.*

INJURIES.	Total.	Relieved.		Not Relieved.		Died.		In hosp. 31st Dec., 1890.	
		M.	F.	M.	F.	M.	F.	M.	F.
UNCLASSIFIED:									
Opium poisoning	1	...	1	...	...	...	...	...	...
Alcoholic collapse	1	1	...	...	...	...	...	...	...
INJURIES:									
Extensive burn	1	...	...	...	...	...	1	...	...
Local burn	11	2	3	...	...	1	2	1	2
Extensive scald	2	1	1	...	...	...	...	...	...
Local scald	7	3	4	...	...	...	...	...	...
Immersion(attempted suicide)	5	2	3	...	...	...	...	...	...
Accidental poisoning	1	...	1	...	...	...	...	...	...
Injuries of head:									
Contusion	2	2	...	...	...	...	...	...	...
Scalp wound	21	11	9	...	...	...	...	...	1
Local destruction of scalp by corrosive fluid	1	1	...	...	...	...	...	...	...
Bullet wound of scalp	1	1	...	...	...	...	...	...	...
Fractured vertex	4	1	1	...	...	2	...	...	...
„ base	2	2	...	...	...	...	...	...	...
Concussion of brain	29	20	8	...	...	...	...	1	...
Laceration of brain	1	...	...	...	...	1	...	...	...
Injuries of face:									
Contusion	4	2	2	...	...	...	...	...	...
Contused wound	2	1	1	...	...	...	...	...	...
Incised wound	3	2	1	...	...	...	...	...	...
Fractured inferior maxilla	1	1	...	...	...	...	...	...	...
Subluxation of inferior maxilla	1	...	...	...	...	...	...	1	...
Injuries of ear:									
Lacerated wound	1	1	...	...	...	...	...	...	...
Injuries of eye and appendages:									
Ruptured eyeball	3	3	...	...	...	...	...	...	...
Punctured wound of eyeball	1	1	...	...	...	...	...	...	...
Wound of cornea	1	...	1	...	...	...	...	...	...
Contused wound of eyelid	3	3	...	...	...	...	...	...	...
Injuries of neck:									
Cut throat (suicidal)	1	1	...	...	...	...	...	...	...
Lacerated wound	1	1	...	...	...	...	...	...	...
Injuries of back:									
Fractured spine	1	...	...	...	...	1	...	...	...
Contusions	13	9	3	...	...	...	...	1	...
Injuries of chest:									
Contusions	3	2	1	...	...	...	...	...	...
Fractured ribs	4	3	...	...	...	1	...	...	...

TABLE I.—*continued.*

INJURIES.	Total.	Relieved.		Not Relieved.		Died.		In hosp. 31st Dec., 1890.	
		M.	F.	M.	F.	M.	F.	M.	F.
INJURIES—*continued.*									
Injuries of abdomen:									
Contusion	1	1	...	...	...	...	...	...	...
Blow in epigastrium—collapse	1	1	...	...	...	...	...	...	...
Injuries of pelvis:									
Fracture of iliac crest	1	1	...	...	...	...	...	...	...
Injuries of upper limb:									
Contusions	4	3	1	...	...	...	...	...	...
Incised wound	4	3	...	...	...	...	...	1	...
Lacerated wound	1	1	...	...	...	...	...	...	...
Subcoracoid dislocation of humerus	3	2	...	...	...	...	1	...	...
Dislocation of finger	1	1	...	...	...	...	...	...	...
Crushed arm	1	1	...	...	...	...	...	...	...
„ hand	4	4	...	...	...	...	...	...	...
„ fingers	14	13	...	...	...	...	...	1	...
Needle in shoulder	1	...	...	1	...	...	...	...	...
Simple fracture:									
Clavicle	1	1	...	...	...	...	...	...	...
Humerus (extra-capsular)	1	1	...	...	...	...	...	...	...
„ (shaft)	2	1	...	...	...	1	...	...	...
Radius	2	...	2	...	...	...	...	...	...
Ulna	1	...	1	...	...	...	...	...	...
Radius and ulna	1	1	...	...	...	...	...	...	...
Compound fracture:									
Humerus	1	...	...	...	...	...	...	1	...
Ulna	1	1	...	...	...	...	...	...	...
Radius and ulna	1	...	1	...	...	...	...	...	...
Injuries of lower limb:									
Contusions	14	10	4	...	...	...	...	...	...
Lacerated wound	5	3	...	...	...	...	...	1	1
Incised „	2	2	...	...	...	...	...	...	...
Contused „	3	2	1	...	...	...	...	...	...
Gun-shot wound of ankle	1	1	...	...	...	...	...	...	...
Sprained ankle	16	9	6	...	...	...	...	1	...
Crushed foot	1	...	1	...	...	...	...	...	...
Needle in knee	1	1	...	...	...	...	...	...	...
„ in leg	1	...	1	...	...	...	...	...	...
„ in foot	1	1	...	...	...	...	...	...	...
Traumatic synovitis of knee joint	3	2	1	...	...	...	...	...	...
Acute spreading traumatic gangrene	1	...	...	...	...	1	...	...	...
Old dislocation of femur	1	1	...	...	...	...	...	...	...
Simple fracture:									
Femur (intracapsular)	1	...	1	...	...	...	...	...	...
„ (extracapsular)	3	2	1	...	...	...	...	...	...
„ (shaft)	15	10	2	...	...	...	...	2	1

TABLE I.—*continued.*

INJURIES.	Total.	Relieved.		Not Relieved.		Died.		In hosp. 31st Dec., 1890.	
		M.	F.	M.	F.	M.	F.	M.	F.
INJURIES—*continued.*									
Patella	3	1	1	...	...	...	...	1	...
Tibia	6	4	2	...	...	...	...	...	...
Fibula	4	3	...	...	...	...	...	1	...
Tibia and fibula	12	9	2	...	...	...	...	1	...
Pott's	9	7	1	...	...	...	...	1	...
External malleolus	1	1	...	...	...	...	...	...	...
Toes	3	2	...	...	...	...	...	...	1
Compound fracture:									
Tibia and fibula	1	1	...	...	...	...	...	...	...
Tibia	2	1	...	...	...	...	...	1	...
UNCLASSIFIED:									
"Run over"—Shock	3	3	...	...	...	...	...	...	...
Dog-bite	1	...	1	...	...	...	...	...	...
Hairpin in urethra	1	1	...	...	...	...	...	...	...

TABLE II.—*Operations.*

OPERATIONS.	Total.	Discharged. M.	Discharged. F.	Died. M.	Died. F.	In hosp., 31st Dec., 1890. M.	In hosp., 31st Dec., 1890. F.	Remarks.
HEAD AND NECK:								
For depressed fracture of skull	2	...	1	1	...	...	...	Trephining.
„ abscess of scalp	2	...	2	...	...	...	...	
„ sebaceous cyst of face	1	1	...	...	...	...	...	Excision.
„ „ „ neck	1	1	...	...	...	...	...	„
„ dermoid cyst of face	1	1	...	...	...	...	...	„
„ „ under chin	1	...	1	...	...	...	...	„
„ epithelioma of lip	3	3	...	...	...	...	...	„
„ „ tongue	3	2	1	...	...	...	...	2 removal of portion, 1 removal of whole.
„ recurrent epithelioma in cervical glands	1	...	1	...	...	...	...	Removal.
„ rodent ulcer of face	1	...	1	...	...	...	...	Excision.
„ keloid	1	...	1	...	...	...	...	
„ nævus of upper lip	1	1	...	...	...	...	...	Ligatured.
„ nævus of face	3	2	1	...	...	...	...	1 cautery, 1 electrolysis, 1 excision.
„ cleft palate	3	2	1	...	...	...	...	1 soft palate only.
„ necrosis of superior maxilla	2	...	2	...	...	...	...	Sequestrotomy.
„ necrosis of inferior maxilla	2	1	1	...	...	...	...	„
„ cyst of superior maxilla	1	...	1	...	...	...	...	Opened and drained.
„ fractured inferior „	1	1	...	...	...	...	...	Wiring.
„ lupus of face	3	2	1	...	...	...	...	Scraping.
„ adenoid vegetations of naso-pharynx	11	7	4	...	...	...	...	Removal with forceps and curette.
„ strumous glands in neck	8	3	5	...	...	...	...	3 excision, 5 incision and scraping.
„ aneurysm of subclavian artery	1	...	...	1	...	...	...	Ligature of innominate artery. Death from pneumonia.
„ mastoid abscess	2	2	...	...	...	...	...	Trephining mastoid cells
„ otitis media	1	1	...	...	...	...	...	Scraping.
„ abscess of thyroid gland	2	2	...	...	...	...	...	Incision and draining.
Tracheotomy	3	1	...	...	2*	...	...	1 for pressure by malignant glands in neck, *1 for œdema of larynx due to epithelioma of œsophagus, *1 for pressure by enlarged thyroid (exophthalmic goitre).
EYES:								
For cataract	10	3	7	...	...	...	...	6 needling, 3 extraction.
„ strabismus	2	1	1	...	...	...	...	
„ ectropion	2	...	2	...	...	...	...	1 transplantation of skin flap, 1 removal of edge of conjunctiva.

TABLE II.—*continued.*

OPERATIONS.	Total.	Discharged.		Died.		In hosp., 31st Dec., 1890.		Remarks.
		M.	F.	M.	F.	M.	F.	
EYES—*continued.*								
For entropion	1	1	...	...	...	...	...	Removal of piece of skin.
Excision of globe	11	7	4	...	...	...	...	
Iridectomy	8	4	4	...	...	...	...	
THORACIC REGION:								
For empyema	2	1	...	1	...	...	...	Drainage.
„ lipoma of back	1	1	...	...	...	...	...	Removal.
„ enlarged axillary gland	1	...	1	...	...	...	...	Excision.
„ fibro-adenoma of breast	5	...	5	...	...	...	...	Excision of growth.
„ cyst of breast	1	...	1	...	...	...	...	Excision of cyst wall.
„ scirrhus of breast	9	1	6	...	2*	...	...	Excision of breast, *1 from bronchitis, 1 from pyæmia.
„ recurrent scirrhus of breast	5	...	5	...	...	...	...	Excision of growth.
„ abscess and (?) chronic mastitis	1	...	1	...	...	...	...	Excision of breast.
„ tubercular axillary glands	3	1	2	...	...	...	...	Excision.
„ abscess in chest wall	2	2	...	...	...	...	...	Opening and draining.
ABDOMINAL REGION:								
For radical cure of inguinal hernia	4	4	...	...	...	...	...	
„ strangulated inguinal hernia	3	2	...	...	1*	...	...	*On 2nd day from perforation and peritonitis (child 14 months old).
„ strangulated umbilical hernia	1	...	1	...	...	...	...	
„ strangulated femoral hernia	5	...	...	...	5*	...	...	*No. 1. On 9th day after operation from peritonitis. Sac was unopened and gut quite healthy. No 2. On 9th day after operation from morbus cordis. Suppuration of wound and suppurative parotitis. No peritonitis. No. 3. On 6th day after operation from morbus cordis. Fæcal fistula formed. No peritonitis. No. 4. On 12th day after operation from bronchitis and emphysema. Fæcal fistula formed. No peritonitis. Old pelvic abscess communicating with bladder. No. 5. On 5th day after operation from phthisis and bronchitis. Small portion of ileum of a dark colour. Peritonitis confined to this part.

TABLE II.—*continued.*

OPERATIONS.	Total.	Discharged.		Died.		In hosp., 31st Dec., 1890.		Remarks.
		M.	F.	M.	F.	M.	F.	
ABDOMINAL REGION—*cont.*								
For renal calculus	1	...	1	...	...	...	...	Nephro-lithotomy.
„ „ „ and purulent pyelo-nephritis	1	...	1	...	...	...	...	Nephrectomy (old nephro-lithotomy case).
„ peri-renal abscess	1	...	1	...	...	...	...	Opened and drained.
„ internal strangulation by band of adhesion.	1	...	...	...	1	...	...	Abdominal section and enterectomy for gangrene.
„ intestinal obstruction	2	...	...	...	2	...	...	1 abdominal section and left colotomy (malignant of sigmoid flexure). 1 right colotomy. ? Malignant disease of colon. (No P.M.)
„ fæcal fistula	1	...	...	1	...	...	...	Formation of artificial anus. Death from perforation and peritonitis.
ABOUT RECTUM AND ANUS:								
For hæmorrhoids	12	7	5	...	...	...	...	
„ fistula	11	8	3	...	...	...	...	
„ fissure	1	1	...	...	...	...	...	
„ stricture of rectum	2	2	...	...	...	...	...	1 dilated, 1 colotomy.
„ myxomatous polypus of rectum	1	1	...	...	...	...	...	Abscission.
„ carcinoma of rectum	1	...	...	1	...	...	...	Excision of rectum. Death 4 days after operation from suppurative pelvic cellulitis.
„ ischio-rectal abscess	3	3	...	...	...	...	...	
ON THE GENITO-URINARY ORGANS:								
For stricture of urethra	4	4	...	...	...	...	...	Dilatation.
„ extravasation of urine	1	...	...	1	...	...	...	Perineal section.
„ phimosis	26	26	...	...	...	...	...	Circumcision.
„ paraphimosis	5	5	...	...	...	...	...	2 circumcision.
„ venereal warts	3	1	2	...	...	...	...	
„ urethral caruncle	1	...	1	...	...	...	...	
„ vesical calculus	4	4	...	...	...	...	...	Lithrotity.
„ villous growth in bladder	1	...	1	...	...	...	...	Removal by ecraseur after rapid dilatation and incision of urethra.
„ varicocele	7	7	...	...	...	...	...	Ligature and excision of veins.
„ hydrocele	2	2	...	...	...	...	...	Injected with tr. iodine.
„ undescended testicle	1	1	...	...	...	...	...	
„ tubercular testis	1	1	...	...	...	...	...	Castration.
„ lacerated perineum	1	...	1	...	...	...	...	

TABLE II.—*continued.*

OPERATIONS.	Total.	Discharged.		Died.		In hosp., 31st Dec., 1890.		Remarks.
		M.	F.	M.	F.	M.	F.	
ON UPPER LIMB:								
For fibrous ankylosis of shoulder	3	2	1	...	...	...	...	Breaking down adhesions.
For dislocation of shoulder (old)	1	...	...	...	1	...	...	Incision, division of adhesions and reduction. Death from bronchitis on 6th day.
„ necrosis of humerus ...	1	1	...	...	...	...	...	
„ badly set fractured ulna	1	...	1	...	...	...	...	Refracture.
„ ostitis of radius	1	...	1	...	...	...	...	Linear osteotomy.
„ necrosis of olecranon process	1	1	...	...	...	...	...	Sequestrotomy.
„ tubercular disease of wrist joint	1	1	...	...	...	...	...	Excision of joint.
„ ganglion of wrist.....	4	2	2	...	...	...	...	2 excision, 2 incision and scraping.
„ Dupuytren's contraction	2	2	...	...	...	...	...	Subcutaneous division of fascia.
„ contracted fingers	1	1	...	...	...	...	...	Forcible straightening.
„ webbed finger	1	1	...	...	...	...	...	
„ necrosis of 5th metacarpal	1	1	...	...	...	...	...	Excision.
ON LOWER LIMB:—								
For housemaid's knee	5	...	5	...	...	...	...	4 opening and draining, 1 excision of bursa.
„ genu extrorsum	1	...	1	...	...	...	...	Osteotomy of femora.
„ genu valgum	2	2	...	...	...	...	...	1 Macewen's operation, 1 osteotomy of tibiæ.
„ talipes equinus	5	2	3	...	...	...	...	4 tenotomy of tendo achilles.
„ „ valgus	2	...	2	...	...	...	...	1 removal of portion of scaphoid, 1 excision of wedge from tarsus.
„ „ equino-varus ...	6	2	4	...	...	...	...	3 excision of wedge from tarsus, 1 tenotomy.
„ „ equino-valgus...	2	1	1	...	...	...	...	1 removal of portion of scaphoid, 1 division of tendo achilles.
„ pes planus	4	4	...	...	...	...	...	"Firing."
„ morbus coxæ.....	4	4	...	...	...	...	...	3 incision and drainage, 1 excision of head of femur.
„ contracted hip.....	4	3	1	...	...	...	...	1 subcutaneous division of bands, 3 breaking down adhesions.
„ pulpy knee	1	1	...	...	...	...	...	Excision of joint.
„ suppuration of knee joint	7	5	2	...	...	...	...	1 excision, 6 opening and draining.
„ old dislocation of hip...	1	1	...	...	...	...	...	Excision of head of femur.
„ congenital cyst of buttock.....	1	1	...	...	...	...	...	Excision.

TABLE II.—*continued.*

OPERATIONS.	Total.	Discharged.		Died.		In hosp., 31st Dec., 1890.		Remarks.
		M.	F.	M.	F.	M.	F.	
ON LOWER LIMB—*cont.*								
For aneurysm in scarpa's triangle	1	1	...	...	...	...	...	Ligature of external iliac artery.
„ sciatica	3	2	1	...	...	...	...	Stretching sciatic nerve.
„ enlarged semimembranosus bursa	1	...	1	...	...	...	...	Excision.
„ varicose veins	5	1	2	...	...	...	...	2 excision, 1 acupressure.
„ necrosis of femur	4	3	1	...	...	...	...	Sequestrotomy.
„ „ tibia	6	5	1	...	...	...	...	„
„ caries of tarsus	11	4	7	...	...	...	...	Opening abscess, gouging, etc., 1 excision of os calcis.
„ ingrowing toe-nail	2	2	...	...	...	...	...	Avulsion.
AMPUTATIONS:								
Of fingers	20	18	2	...	...	...	...	
„ toes	7	7	...	...	...	...	...	
Through arm	1	1	...	...	...	...	...	For compound comminuted fracture of humerus.
Syme's amputation	2	1	1	...	...	...	...	For caries of tarsus.
Through middle third of leg	1	...	1	...	...	...	...	For senile gangrene.
„ upper „	1	...	1	...	...	...	...	For necrosis of tibia after compound comminuted fracture.
„ knee joint	1	...	1	...	...	...	...	For suppurative arthritis.
„ thigh	6	4	2	...	...	...	...	1 for suppurative arthritis of knee joint, 1 for acute suppurative periostitis of femur, 2 for tubercular arthritis of knee joint, 1 for osteomyelitis of femur after excision of hip joint, 1 for tubercular disease of foot and leg.
Of hip joint	2	2	...	...	...	...	...	For morbus coxæ. (Old excision cases.)

REPORT OF CASES IN "ARDEN WARD"

(FOR DISEASES OF WOMEN)

DURING THE YEAR 1890.

By A. M. GOSSAGE.

Number of patients under treatment, January 1st		11
" " admitted during 1890		101
Total		112
Number of patients discharged during the year:		
Cured or relieved	88	
Unrelieved	9	
	—	97
*Died		6
Under treatment, December 31st, 1890		9
Total		112

The mortality during the year was 5·35 per cent.

* 1 abortion, 1 tubercular peritonitis, 1 ovariotomy, 1 uterine fibroid (after operation), 1 septicæmia and acute endocarditis, 1 puerperal pyæmia.

Cases in "Arden Ward."

Nature of Case.	No. of Case.	Initials.	Age.	Result.	No. in Register.	Remarks, treatment, &c.
Uterus:						
Subinvolution	I	F. P.	39	Cured	O. 93	Curetted. Fungous endometritis.
	II	E. S.	29	Relieved	O. 30	After miscarriage.
	III	L. P.	27	„	N. 230	After miscarriage.
	IV	A. D.	30	„	O. 34	After miscarriage.
Endometritis	I	A. B.	28	„	O. 88	Specific.
Retroflexion	I	T. D.	30	„	N. 64	
	II	C. M.	28	„	N. 119	
	III	E. J.	35	„	N. 168	
	IV	M. K.	22	„	N. 226	
	V	M. S.	32	„	N. 222	Menorrhagia.
	VI	E. F.	51	„	O. 59	
	VII	M. Y.	29	„	O. 169	
Prolapse	I	S. J.	50	„	N. 101	
	II	E. H.	70	Unrelieved	N. 206	? Carcinoma of rectum.
	III	E. W.	29	Relieved	O. 108	Perinæum deficient.
	IV	E. B.	38	„	O. 155	Perinæum deficient.
Fibroid	I	E. M.	46	„	N. 26	Interstitial. Electrolysis, 28 applications of ten minutes each.
	II	M. F.	35	Unrelieved	N. 53	Interstitial. Electrolysis, 6 applications of ten minutes each.
	III	L. L.	40	Relieved	N. 55	Interstitial. Electrolysis, 14 applications of ten minutes each.
	IV	M. C.	37	„	N. 110	Impacted. Raised.
	V	E. C.	35	„	N. 131	Interstitial. Electrolysis, 8 applications of ten minutes each.
	VI	E. G.	51	„	N. 246	Multiple, incarcerated. Sloughing. One large fibroid nearly disappeared. Bronchitis.
	VII	M. R.	55	Death	N. 208	Hysterectomy.
	VIII	H. G.	44	Relieved	O. 112	Multiple, one becoming pediculated and protruding through os.
Malignant Disease	I	E. G.	64	Unrelieved	O. 101	Malignant papilloma from fundus.

CERVIX UTERI :						
Stenosis	I	M. M.	34	Cured	O. 34	Sterility. Divided with metrotome.
	II	M. C.	24	„	O. 104	Sterility. Divided with metrotome. Dysmenorrhœa. Relieved in both cases.
Endocervicitis	I	M. W.	38	Relieved	O. 8	
	II	E. H.	44	„	O. 16	
	III	M. C.	33	„	O. 74	
Syphilitic Ulceration ...	I	E. D.	28	„	N. 59	Improved under specific treatment.
	II	E. M.	37	„	N. 61	Improved under specific treatment.
Polypi.....	I	M. A.	52	Cured	N. 218	Mucous, removed.
	II	S. W.	49	„	O. 120	Multiple mucous, removed by torsion.
	III	E. G.	45	„	O. 129	Multiple mucous, removed by torsion.
Supra Vaginal Hypertrophy, with Procidentia Uteri	I	J. B.	30	„	N. 97	Cervix amputated with galvano-cautery.
Rodent Ulcer.....	I	L. D.	47	Relieved	N. 180	Cautery.
Malignant Disease.....	I	G. W.	32	„	N. 70	Carcinoma. Operation refused.
	II	A. D.	25	„	N. 85	Epithelioma. Scraped.
	III	M. B.	33	„	N. 110	Carcinoma. Scraped. Cautery.
	IV	E. H.	44	„	N. 123	Carcinoma. Scraped. Cautery.
	V	M. B.	35	„	N. 129 and N. 215	Carcinoma. Scraped. Recurred 2½ months after. Scraped.
	VI	E. C.	42	„	N. 249	Epithelioma. Scraped. Cautery.
	VII	M. H.	47	Unrelieved	O. 18	Carcinoma, affecting also vagina.
	VIII	A. D.	30	„	O. 153	Carcinoma. Operation refused.
OVARY :						
Ovaritis	I	E. F.	25	Relieved	N. 157	Prolapsed and enlarged ovary.
	II	E. B.	24	„	N. 188	Prolapsed and enlarged ovary.
	III.	G. S.	28	„	O. 70	Prolapsed and enlarged ovary.
	IV	F. D.	30	„	O. 162	Prolapsed and enlarged ovary. ? Cystic.
Tumours—						
Cystic.....	I	M. H.	31	Death	N. 236	Multilocular cyst of right ovary. Peritonitis after ovariotomy.
	II	M. F.	16	Cured	O. 158	Dermoid cyst of right ovary. Ovariotomy. Recovery.

CASES IN "ARDEN WARD"—*continued.*

Nature of Case.	No. of Case.	Initials.	Age.	Result.	No. in Register.	Remarks, treatment, &c.
VAGINA:						
Redundant Mucosa	I	A. P.	28	Cured	N. 108	Removed by knife and cautery.
Cystocele	I	E. M.	66	Relieved.	N. 194	
Vaginismus	I	M. J.	22	„	N. 95A	
Vaginal Thrombus	I	A. H.	30	Cured	N. 95	
Cyst of Bartholin's Gland	I	C. G.	31	„	N. 83	Removed. Left gland.
	II	A. P.	17	„	N. 115	Removed. Right gland.
	III	J. L.	25	„	N. 127	Removed. Right gland.
	IV	L. H.	32	„	N. 234	Removed. Right gland.
	V	S. C.	44	„	O. 80	Opened and plugged. Left gland.
Abscess of Bartholin's Gland	I	R. W.	26	„	O. 56	Opened and plugged. Left gland.
VULVA:						
Cyst of Labium	I	E. W.	26	„	N. 200	Removed. Sebaceous cyst.
Epithelioma	I	F. O.	46	Relieved	O. 52	Removed. Villous epithelioma of clitoris and labia minora.
BLADDER, URETHRA, &c.:						
Cystitis	I	S. R.	23	„	O. 41	Subacute.
	II	A. A.	25	„	O. 97	Subacute.
Vascula Caruncle	I	J. G.	48	Cured	N. 165	
	II	E. A.	60	„	N. 220	
	III	J. N.	47	Relieved	O. 1	
DISORDERS OF MENSTRUATION:						
Menopause.....	I	S. L.	36	Recovery	N. 72	
	II	M. C.	52	Relieved	N. 228	Menorrhagia.

Disorders during and after Pregnancy:						
Pregnancy	I	A. S.	38	„	N. 149	Threatened abortion.
	II	E. W.	28	„	N. 188	Contracted pelvis. Premature labour. Child delivered with forceps. Child still-born.
	III	E. S.	20	„	O. 106	Traumatic epilepsy. Since æt 14. Fits and pains in head since admission. Some optic neuritis.
	IV	F. P.	22	„	O. 143	Albuminuria. Œdema of extremities and ascites. Induction of labour at 8th month. Female child living.
Ruptured Perinæum ...	I	E. D.	33	„	N. 196	
	II	H. G.	32	„	O. 77	
Sequelæ of Abortion ...	I	J. B.	42	„	O. 37	
	II	E. H.	27	„	O. 125	
	III	J. A.	28	„	O. 46	
Abortion.....	I	R. M.	38	Death	N. 161	
Puerperal Pyæmia	I	J. O'C.	26	„	O. 28	Confined two weeks before admission. Placenta retained. Removed by hand.
Pelvis:						
Pelvic Peritonitis.....	I	S. P.	20	Relieved	N. 173	Acute. Chill at period.
	II	A. G.	33	„	O. 91	Chronic. Chill at period.
Pelvic Cellulitis	I	A. S.	28	„	N. 138	Chronic. Chill at period.
	II	J. P.	25	„	N. 224	Acute. Abortion.
	III	A. M.	30	„	O. 84	Acute. Chill at period.
	IV	H. W.	40	„	O. 140	Acute. Puerperal. Pelvic abscess. Opened in right groin.
	V	C. B.	39	„	O. 149	Chronic. Chill at period.
Pelvic Hæmatocele	I	A. G.	29	„	N. 49	
	II	M. S.	21	Recovery	N. 143	Acute rheumatism.
	III	E. G.	30	„	N. 176	? Tubal pregnancy.
Unclassified:						
Tubercular Peritonitis...	I	A. P.	17	Death	N. 153	
Abdominal Tumour.....	I	R. T.	70	Relieved	N. 202	? Renal tumour. Chronic bronchitis.
	II	A. B.	26	Unrelieved	O. 131	Exploratory abdominal incision. Tumour post-peritoneal. Not removable.
Thrombus of internal saphena vein.....	I	C. J.	34	Relieved	O. 26	Left.
Acute endocarditis	I	R. J.	42	Death	O. 4	? Septicæmia after dilatation of cervix and removal of small polypus.

THE GUTHRIE SOCIETY.—STATEMENT FOR 1890-91.

By Mr. BRICE.

The Guthrie Society has now completed its fourth year, and continues to be a success. Its financial affairs are in a flourishing condition, and fresh members are constantly being enrolled, which more than compensates for the few who annually retire on account of their becoming qualified, and leaving Westminster behind them. It is gratifying to notice that many gentlemen become life-members before they leave.

The usual nine Meetings of the Society have been held during the year, the total number of attendances amounting to 280, averaging 31 for each Meeting, which, as compared with last year, shows a slight increase.

It is to be noted, with great regret, that the attendance of the members of the staff is becoming smaller; in fact, that, with one or two well-known exceptions, none of them ever turn up. It is generally felt that the presence of the members of the staff adds to the importance of the Meetings, and makes the discussions more brilliant. It is, therefore, hoped in the future that they will be more often seen at the Meetings.

The Society now numbers 106, 20 new members having been enrolled during the year.

The following are some of the subjects brought before the Monthly Meetings of the Society:—

A case of the persistence of the branchial clefts of the neck was shown by Mr. C. Stonham.

A Paper on 'The Island of Atlantis,' by Dr. Mercier, which was followed by a Demonstration of Histological and Pathological Specimens.

A Report on three cases of fracture of the skull, by Mr. A. W. Harrison.

A Paper on 'The Relation of Medicine and Surgery to Dentistry,' by Mr. Glassington.

Exhibition of Surgical Instruments, lent by Messrs. Wright and Co., and demonstrated by Mr. Montague.

A Paper on 'Some Objections to Darwinism' (Part I.), by Mr. W. G. Spencer, followed by a Demonstration of Pathological Specimens.

A Paper on 'Some Objections to Darwinism' (Part II.), by Mr. W. G. Spencer, followed by a Paper on 'Lectures, their Use and Abuse,' by Mr. H. Wolseley-Lewis.

A Discussion on how the available funds of the Society should be expended.

A Paper on 'Modern Explosives and the new Magazine Rifle,' profusely illustrated by experiments, by Dr. Dupré, F.R.S.

A Discussion on Medical Degrees for London Medical Students, opened by Mr. E. Cullinan.

A Paper on, and exhibition of, 'Birds' Nests and Birds' Eggs,' by Mr. C. Stonham.

The Annual General Meeting was held on July 16th, 1891, when a Presidential Address was delivered by Mr. C. Stonham. The Discussion as to how the available funds of the Society should be expended, resulted in the purchase of a microscope.

Some of the Meetings were followed by a Smoking Concert, and it is observed that the attendance is better on these occasions and when papers of general interest are read, than when papers on more professional subjects are brought before the Society.

The Paper by Mr. H. Wolseley-Lewis on 'Lectures, their Use and Abuse,' and the debate on 'Degrees for London Medical Students,' which was opened by Mr. Cullinan, both proved to be very successful evenings, and a larger number of students spoke at these Meetings than at any of the others held by the Society. There is, therefore, now under consideration in the Committee, the advisability of occasionally holding debates on subjects other than medical and scientific at the Monthly Meetings.

Dr. Abraham has presented the Society with a large number of histological specimens.

At the Annual General Meeting, in July, Dr. Allchin was elected President, in the place of Mr. C. Stonham; Mr. P. Macleod Yearsley was elected Vice-President; and Mr. A. Percy Brice, Honorary Secretary, on the retirement of Mr. H. A. Stonham.

November 19th, 1891.